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Panic attack symptoms in patients with diabetic peripheral neuropathy

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ABSTRACT

Assessment of diabetic peripheral neuropathy (DPN) usually focuses on nerve damage resulting from hyperglycaemia. However, screening for common psychiatric disorders may improve the recognition of psychopathology in patients with DPN.

This epidemiological cohort study aimed to evaluate the prevalence of panic attack symptoms in patients with DPN compared toa control group of healthy individuals without type 2 diabetes mellitusorDPN.Additionally, this study sought to compare the severity of these symptoms between the two groups.The study was conducted via a survey over three years in an accredited practice of physicians at the Medical University-Sofia.

A total of267 participants were included, comprising 83 patients with DPN and 184healthy controls. Both groups completed the Prime-MD Patient Health Questionnaire. The results indicated significant differences between the two study groups(t[127.513] = 3.293; $p < 0.01$), and patients with DPN had a higher prevalence of panic attack symptoms than those in the control group.Furthermore, significant differences were observed in the severity of panic attack symptoms within the DPN group (t[(81] = 2.017, $p < 0.05$).Patients who had experienced DPN for more than one year reported more severe symptoms than those who had experienced it for less than oneyear. Our results indicate that the high prevalence of panic attack symptoms inpatients with DPN highlights the need for integrated screening for psychiatric disorders within the overall management plan for diabetes mellitus.

1. Introduction

Anxiety disorders are the most common mental disorders worldwide [1]. Epidemiological studies have shown that the clinical manifestation of panic attack (PA) symptoms is associated with an increased risk of developing anxiety and mood disorders [2].PA symptoms occurring outside of a panic disorder (PD) predict important clinical outcomes and indicate severe psychopathology across a wide range of mental disorders [3].Therefore, the assessment of PA symptoms is performed as a separate dimension of all mental disorders.The high health care costs resulting from not recognizing patients experiencingPA symptoms constitute an economic burden [4].

The assessment of PA symptoms may be particularly important for patients with type 2 diabetes mellitus (T2DM) and diabetic peripheral neuropathy(DPN). Mental health factors could further exacerbate the burden for these patients who already experience poor physical performance and sensory limitations that affect daily activities, reduced quality of life and impair overall functioning [5,6].Literature on psychiatric comorbidities in patients with T2DM and DPN remains scarce [7] and several studies have reported a gap in the assessment of anxiety disorders in patients with T2DM [8,9].

The global epidemic of prediabetes and diabetes has led to an increased prevalenceofDPN, the primary microvascular complication of T2DM [12].DPN and may already be present at the time of T2DM diagnosis, with a prevalence ranging from 26 % to 50 % [10]. DPN is characterized by neurodegeneration of the distal terminals of the first sensory neurones in the dorsal root ganglia and is associated with increased sensitivity and spontaneous activity [11]. These finding suggests that primary afferent hyperexcitability is a critical pathophysiological driver of DPN [12]. In the spinal cord, the increased synaptic transmission of second-order neurones is enhanced by the spatial and temporal summation of a series of nociceptive inputs [13]. This results in increased synaptic transmission in the spinal cord and further amplification of nociceptive signaling through central sensitization [14], which is characterized by a functional shift in the somatosensory system from high-to low-threshold nociception. This may be

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compounded by reduced inhibition of nociceptive neurones by neurotransmitters such as gamma-aminobutyric acid (GABA), serotonin, and norepinephrine in both spinal and supraspinal structures [15,16].

Research on patientswith DPN has demonstrated reduced GABAergic inhibition of nociceptive neurons in the thalamusand found that low GABA levels correlating with neuropathy severity [17].Several studies have implicated dysregulation of top-down connections in the prefrontal cortex in both anxiety and nociception [18]. The prefrontal cortex is connected to the thalamus and limbic cortex and interacts with the amygdala and autonomic nuclei of the brainstem [19], which underlie emotional and physiological responses, thereby regulating anxiety and fear.

Brainstem nuclei, including the noradrenergic locus coeruleus, serotonergic dorsal raphe nucleus, and dopaminergic ventral tegmental area, are the primary source of monoaminergic ascending fibers [20].

The dorsal raphe nucleusisa major source of serotonin in the brainstem and is associated with the pathogenesis of anxiety, panic, and chronic pain [21]. Evidence suggests that the dorsal raphe nucleus is reciprocally coupled with the locus coeruleus and amygdala [22]. Furthermore, the prefrontal cortex fails to provide top-down inhibitory input to the amygdala, resulting in excessive amygdala activation and unnecessary activation of the entire fear network, which contributes to anxiety and panic [23].

Neuroanatomical studies have established that a series of inputs and outputs from the amygdala are directly related to the pathogenesis of PD [24,25].

1.1. Hypotheses

Hypothesis 1. The prevalence of PA symptoms will be higher in patients with DPN than in healthy controls.

Hypothesis 2. The frequency and severity of PA symptoms will be higher in patients with DPN more than one year before participating in the study than in those who had been diagnosed less than one year before.

2. Materials and methods

This cohort study was conducted over three years (2015–2018) at MEDIK-28 DRUJBA Ltd., an accredited practice for the practical training of physicians and students in the Department of General Medicine at the Medical University-Sofia. Ambulatory records included in the electronic practice database were used as additional source of information. The Primary Care Assessment of Mental Disorders (PRIME-MD [26]) is an instrument for diagnosing depressive and anxiety disorders in general medical practice introduced in 1994 and has been substantially validated over the years. The purpose of the instrument was to identify common psychiatric disorders in the study cohort and enable a rapid screening method for diagnostic orientation.Both the groups completed the Prime-MD Patient Health Questionnaire (PHQ-9). The subscale contains 11 statements measuring PA symptoms within the last 4 weeks. The assessment scale was dichotomous (“yes” or “no”).

2.1. Participants

This study included a sample of 267 participants. Of these, 83 had diabetic neuropathy(35 men and 48 women). The remaining 184 healthy participants comprised the control group (121 men and 63 women). The medical records of the respondents did not include data on concomitant or previous diseases, and patientswith DPN were diagnosed after routine annual screening. Patient evaluation included careful history and clinical examination of the feet. Screening for DPN was conducted by evaluating sensitivity to the pinprick test, sense of touch and pressure with a 10 g monofilament, vibration sensation (128 Hz tuning fork), and ankle reflexes.

Patients were included if they:1) provided informed consent, 2)were aged28-83 years, 3) had T2DM complicated by DPN. Patients were excluded if they had any of the following: 1) hypoglycaemia, anaemia, or thyrotoxicosis, 2) vitamin B12 deficiency, 3) a chronic disease such as heart, kidney, respiratory, orliver failure, 4) neuropathy of another origin, 5) alcohol or drugabuse,6) bipolar disorder or schizophrenia, or 7) a history of a neurological condition such as epilepsy, Parkinson’s disease, Alzheimer’s disease, or another form of dementia.Healthy control participants were randomly selected during their annual preventive examination visits. Medical records did not provide any data regarding concomitant or previous diseases.

2.2. Data analysis

We conducted an independent-sample *t*-test to evaluate differences in PA experiences between patients with DPN and controls. We then applied another independent sample *t*-test to evaluate differences in the severity of PA symptoms between patients diagnosed with DPN more than one year before data collection and those diagnosed less than one year before data collection. All analyses were performed using IBM SPSS v.28. No missing data were encountered.

3. Results

The sample comprised 267 participants, with ages ranging from 28 to 83 years (M = 65 years; SD = 11.67). The control group consisted of 184 healthy individuals with ages ranging from 19 to 86 years (M = 50.32, SD = 15.93).

Medical records showed that among patients with DPN had concomitant cardiovascular diseases 60 patients (72 %), seven patients (9 %) had neurological comorbidities, one (1 %)had a psychiatric disease and 15(18 %)had no concomitant disease. Thirty-seven patients had previously received treatment from a neurologist or psychiatrist, whereas 46 had received no prior treatment. Furthermore, 37patients were newly diagnosed with diabetes (within the past year)and 46 patients had long-standing diabetes (diagnosed more than a year ago).

One-third of the respondents with DPN reported a rapid or pounding heartbeat (36.1 %); vertigo, dizziness, or faintness (31.3 %); a tingling or burning sensation in any part of the body (31.1 %); and sweating (28.9 %). Over one-quarter of the healthy participants reported experiencing a rapid or pounding heartbeat; sweating (28.8 %); hot flushes or chills (25.5 %); vertigo, dizziness, or faintness (25.0 %); breathlessness (23.4 %); and trembling (22.8 %).Fig. 1shows a full breakdown of the symptoms reported by both groups.

After incorporating statements from the (PHQ-9) on an aggregate scale, we observed that more than half of the respondents with DPN (55.40 %) and healthy controls (55.4 %) did not report experiencing the aforementioned PA symptoms. However, among those with DPN, 20.5 % had moderately severe symptoms and 6 % had severe symptoms, whereas only 4.9 % and 3.8 % of healthy participants reported moderately severe and severe symptoms, respectively (Fig. 2).

A *t*-test of independent samples in a between-subjects design revealed significant differences in the severity of PA symptoms: $t(127.513) = 3.293$; $p < 0.01$, $d = 3.57$, 95 % CI (−2.74; −0.68). The DPN group experienced more severe symptoms (M = 4.13, SD = 4.21) than the healthy group (M = 2.42; SD = 3.24).

Finally, a *t*-test was conducted to examine differences in the prevalence of PA symptoms between patients who had DPN for less than a year and those who had DPN for more than a year:$t(81) = 2.017$, $p < 0.05$, $d = 1.40$, 95 % CI (−1.24; −0.01). PA symptoms were more prevalent in patients diagnosed with DPN for more than one year (M = 2.43, SD = 3.49) than in those diagnosed less than oneyear (M = 1.81, SD = 2.95).

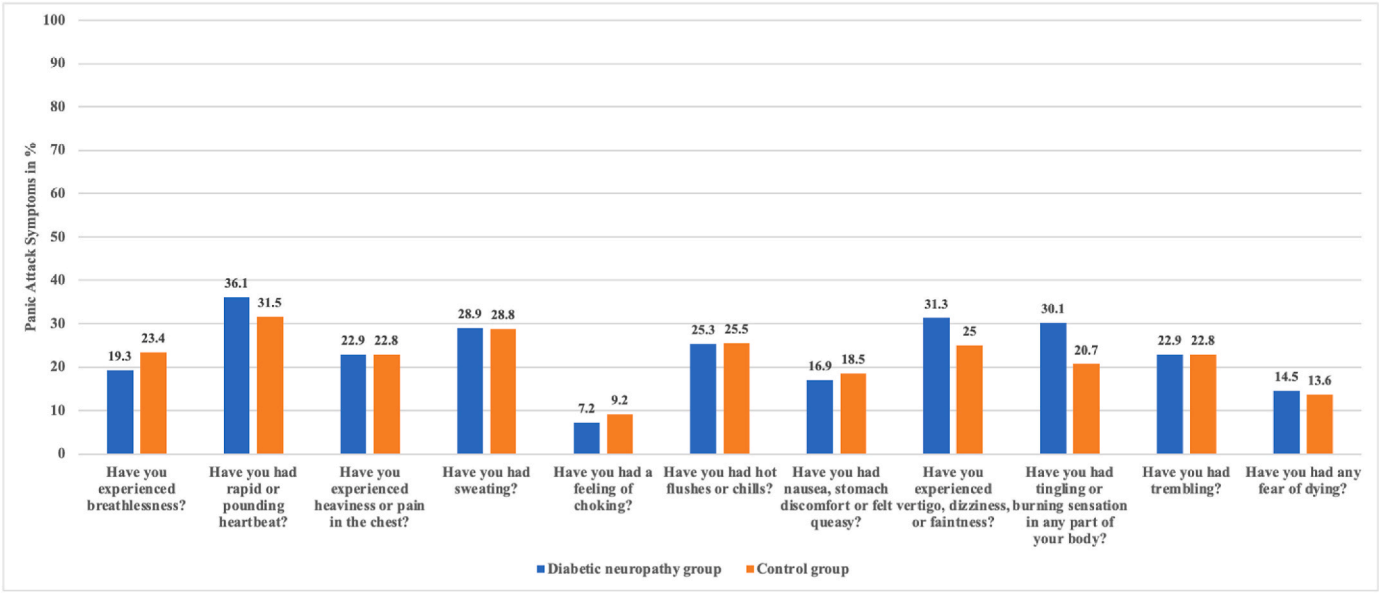

Fig. 1. Distribution of panic attack symptoms.[11]

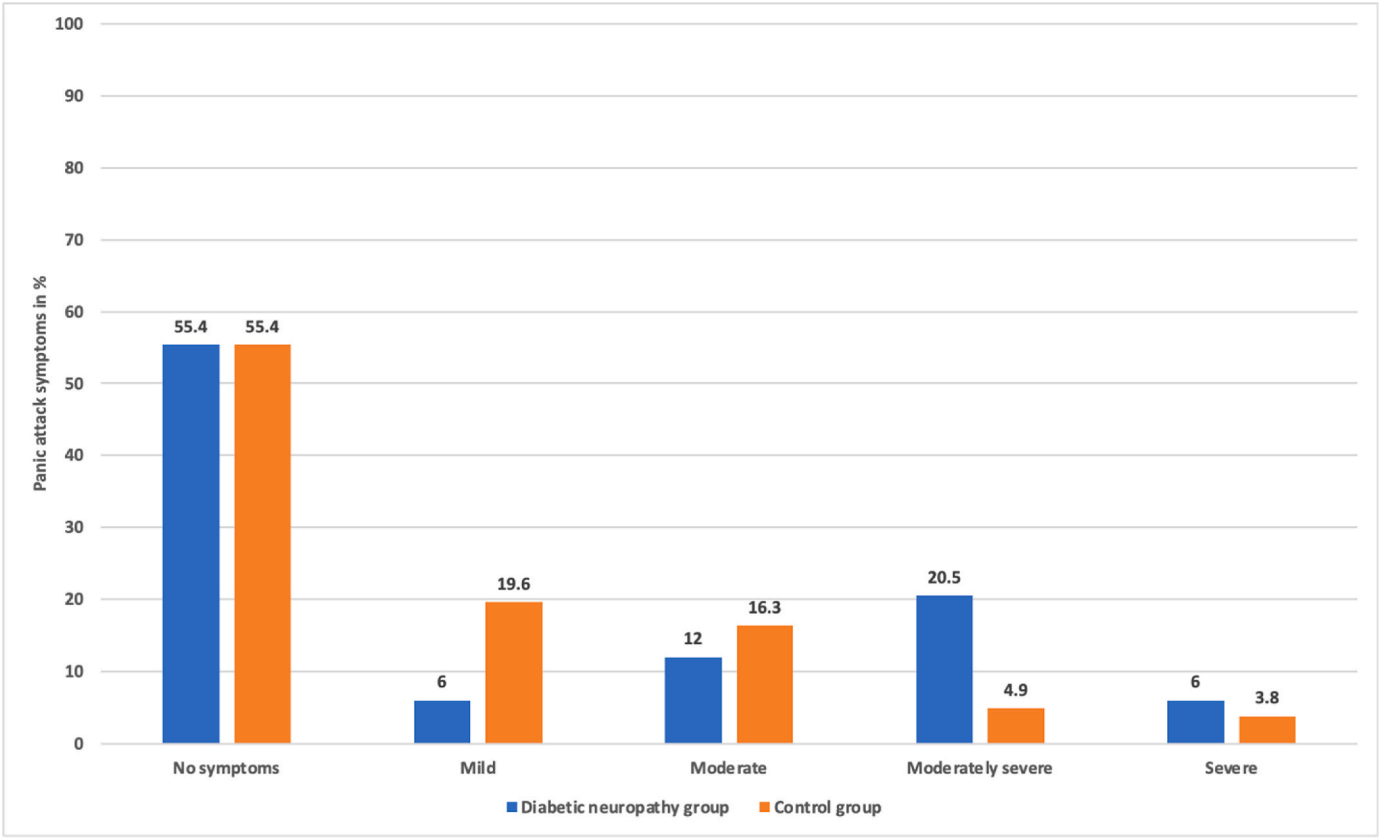

Fig. 2. Distribution by severity of panic attack symptoms.

4. Discussion

Underdiagnosis of anxiety and mood disorders is one of the main challenges faced by individuals with diabetes. Research suggests that PA symptoms are associated with various mental disorders including anxiety, depression, mood disorders, and substance abuse not justPD [27]. Additionally, the assessment of PA symptoms has been found to increase comorbidity and suicide rates while having a negative impact on treatment response in several diseases [28].

Data on the course of T2DM complicated byDPN and the pathophysiological relationship of PA symptoms remain unclear.This study aimed to investigate differences in the prevalence of PA symptoms between patients with DPN and healthy controls. Additionally, we compared the severity of these symptoms between patients who had been diagnosed with DPN more than one year before the study and those who had been diagnosed less than one year before the study.Patients

diagnosed with DPN had a higher prevalence of PA symptoms than the healthy controls. It was also found that as the duration of illness increased, the likelihood of more severe symptoms associated with PA symptoms increased.

Although the onset of PA symptoms in patients with DPN negatively affects diabetes treatment outcomes, its recognition by healthcare providers remains poor. Moreover, the quality of life of these patients is further impaired by a high incidence of anxiety, depression, and sleep disorders. Literature on the prevalence of PD in patients with diabetes reports a frequency ranging from 2.5 % to 5.1 % [29].

Our study confirmed the findings of a previous study [30], which was one of the few published studies with a comparable research design and showed an increased incidence of PA symptoms with a prevalence of 4.4 % in patients with diabetes mellitus. However, that study was conducted in a hospital setting, whereas our study was conducted ina primary healthcare setting. The higher rate of PA symptoms in our study (6 %) was probably due to complication of diabetes with DPN. Another difference is that we also evaluated PA symptoms in a control group without T2DM or DPN, and the observed differences in PA symptoms between patients with DPN and healthy controls may be related to several alternative interpretations of the results. The first possibility is that the clinical manifestation of PA symptoms is associated with higher anxiety levels in patients with T2DM.Another potential explanation is that increased nociceptive afferent inputs to the brain and the process of central sensitization [31]may lead to the occurrence of PA symptoms and anxiety in patients with DPN. The third possible mechanism is the activation of the fear neural network through the amygdala and brainstem nuclei responsible for emotions [32]. Changes in the nociceptive nervous system of patients with DPN are associated with sensory and emotional experiences. Therefore, emotional component of the experience reflects a large and constant increase in nociceptive afferent input involving the corticolimbic system [33]. Therefore, the higher prevalence of PA symptoms in patients with DPN is likely secondarily related to pathoanatomical and pathophysiological processes in the peripheral nervous system.Additionally, with the progression of DPN after the first year of the disease, comorbid psychopathology is established, and manifestations of the disease occur alongside heightened anxiety levels.

4.1. Limitations

Our study has several limitations. First, it was limited by the small number of participants. Therefore, future studies should replicate this study with a larger sample size. Second, the study was conducted in a specific geographic region (Sofia, the capital of Bulgaria), which limits our findings to only a small portion of global population and even limits the possibility of generalisation to the entire Bulgarian population. Future studies should include more diverse samples to identify possible cross-cultural differences.

5. Conclusion

The present study showed significant differences in PA symptoms between patients with DPN and healthy controls. Patients who had T2DM and DPN for more than one year exhibited significantly higher levels of anxiety than those with newly diagnosed diabetes.This study adds to the current understanding of the psychological aspects of anxiety in patients with DPN. These results contribute to the development of a more detailed assessment of the psychophysical condition of patients and suggest improvements in treatment options. When determining treatment, an individualized approach should be applied using similar scales and questionnaires for the rapid screening and diagnosis of anxiety disorder symptoms. The psychometric indicators of the adapted and standardized scale for the Bulgarian sociocultural context for measuring PA symptoms and prevalence can be successfully applied by general practitioners to treat patients with DPN. The existing research gap regarding the brain mechanisms underlying PA manifestation and increased anxiety in patients with DPN presents an opportunity for further studies in this field.

[1] responses are presented in percentages.

CRediT authorship contribution statement

Nayden H. Manolov: Writing – original draft, Investigation, Conceptualization. **Arman Sh Postadzhiyan:** Supervision. **Sonya M. Karabeliova:** Software, Formal analysis, Data curation. **Peter M. Marinov:** Supervision, Methodology, Conceptualization.

Declaration of competing interest

None.

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