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Mini Review

Involvement of trigeminal neuralgia in type 2 diabetes

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Summary

The aim of this paper is to describe orofacial neuralgic pain related to diabetes mellitus pathology with a clinical report of a female patient who suffered from diabetic polyneuropathy. A 61-year-old female patient was treated neurologically and dentally due to suspicions of Trigeminal Neuralgia (TN) and disorders of the temporomandibular joint. Recent symptoms were burning and heat, electric shock sensation related to the right side of the face and particularly the second right premolar tooth. The patient had received regular insulin therapy (type 2 diabetes mellitus) for the last 10 years and was diagnosed with diabetic polyneuropathy with a higher value of glycosylated hemoglobin HbA1c (59 mmol/mol). The psychological evaluation showed an elevated anxiety level according to Spielberger's State-Trait Anxiety Inventory. The most common neurogenic complication of type 2 DM is diabetic polyneuropathy. The functional status of the trigeminal reflex pathways was reflected through the blink reflex. There were a few existing reports of the relationship between diabetes mellitus and TN- related orofacial pain, which is discussed in this paper.

Introduction

Trigeminal Neuralgia (TN) is the most common type of neuropathic pain in the stomatognathic system. It is difficult to determine the prevalence because the disease is often not recognized as neuralgia so the symptomatology is in dental practice initially related to odontogenic pain of unclear etiology. The complete and definitive diagnostics as well as treatment procedures are the responsibility of a neurologist [1-3].

Also, co-morbidity of TN or pain related to TN and many systemic diseases may induce orofacial pains, whether as an accompanying condition to the main disease (metabolic and endocrine diseases, rheumatic diseases, etc.) or identified as a risk factor (for example, hypertension for TN) or trigeminal pain caused by trauma [4].

Type 2 Diabetes Mellitus (DM) is a heterogeneous group of diseases characterized by different degrees of insulin resistance, disorders of insulin function, and/or secretion with an increase of glucose production in the liver by the process of gluconeogenesis [5].

The most common neurogenic complication of type 2 DM is diabetic polyneuropathy, which is a microvascular complication characterized by progressive sensory loss with or without neuropathic pain. Its typical clinical expression is burning pain, electrical shocks associated with paraesthesia or dysesthesia, and additional allodynia. Risks and coexisting factors for diabetic polyneuropathy are older age, long diabetes duration, poor glycaemic control, higher BMI, nicotinism, alcohol consumption, elevated systolic blood pressure, peripheral vascular disease, and hypercholesterolemia [6-12].

DM is a lifelong incurable disease that also causes psychological disturbances in patients regarding their coping with a specific diet, exercise, and regular insulin intake [13,14]. For TN, the importance of the electro-neurophysiological blink reflex procedure lies in the presentation of neurogenic abnormalities that manifest as dysfunction of the trigeminal

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sensory system. Besides, the nociceptive blink reflex procedure is particularly important as a diagnostic instrument for TN-related pain disease [15,16].

Diabetic polyneuropathy includes painful sensations of cranial nerves (the third, fourth, sixth, and seventh cranial nerves) [17-19], while the involvement of the fifth (trigeminal) nerve has been described in several clinical cases [20-26]. Hyperglycaemia was previously diagnosed in as many as 33% of patients with TN according to Hindi, et al. [27].

There is a confirmed correlation between glycemic levels, HbA1c, and the progression of late complications in DM, and the level of HbA1c ≤ 7% is defined as the target of good glycemia regulation [28]. There is a clinical correlation between poorly regulated DM expressed by HbA1c interpretation and the manifestation of pain in the orofacial region [29,30].

The aim of this paper is to review orofacial neuralgic pain pathology related to type 2 DM, with a clinical report of a female patient who suffered from diabetic polyneuropathy.

A case report

A 61-year-old retired female patient was referred to the Removable Prosthodontic Department, School of Dental Medicine, University of Zagreb, for differential diagnostics of suspect orofacial pain related to Temporomandibular Joint (TMJ) disorder or TN. The recent symptoms were limited mouth opening, and an unpleasant disorder of sensation (burning and heat, electric shock sensation) related to the right side of the face (Figure 1), particularly the second right premolar tooth. One of the indications of TMJ disorder during the neurological examination was the hyperextension of the right condyle seen on the x-ray image of the TMJ.

Patient's medical history

The patient had been regularly taking insulin (DM type 2) since 2000, and previously, since 1995 had been using oral hypoglycemic drugs. Distal diabetic polyneuropathy was diagnosed as early as 2000 at a neurological examination (chronic polyneuropathy diabetic) together with a superimposed radicular lesion involving L5 dermatomes (the fifth lumbar spinal nerve) bilaterally (more dextrally) and S1 (the first sacral spinal nerve) dextrally. Even then she had a sensation of numbness in her lower legs and pain in both heels as well as occasional lumbar-sacral pain with propagation in the right hip. The examination showed that rheumatologic symptoms (and cervical, and chronic lumbosacral) were more pronounced together with diabetes-related symptoms. Lasègue test was negative which excludes herniated discs, in a patient with lower back pain mostly located at L5, S1, or S2. Normal values of bone density were determined by densitometry from 2001 and 2004. Her body mass index was elevated (BMI 29.36 kg/m^2). The results of the last diabetological examination prior to a neurological treatment, showed unsuccessful diabetes treatment due to additional, borderline dyslipidemia as well as hypertension (blood pressure was RR 170/80 mm Hg without cardiologic symptoms). The results of laboratory tests are shown in Table 1 [28].

Neurological examination

During a recent neurological examination at a clinic, radiculopathy changes on the level of L5 were confirmed by electromyography. Multi-slice Computerized Tomography (CT) of the brain did not reveal fresh signs of ischemia, hemorrhage, expansive process, or extra-axial collection. Transcranial Doppler sonography and extracranial color Doppler, as well as power Doppler sonography, were within normal ranges regarding the patient's age. As an abnormal blink reflex finding, latency values were shown in Table 2 [17,18].

Patient's history related to orofacial pain

The first orofacial symptoms appeared briefly three years ago when the patient experienced stress due to her husband's hospitalization. Since then, she had experienced problems with the second right premolar tooth, which she wanted to extract due to an odd and unpleasant electric shock sensation, although there was no obvious odontogenic symptomatology (Figures 2,3). Later on, there were no strong or frequent attacks until five months ago when her brother died. At that time, the burning sensation returned but more intensively along with the sensation of heat and electric shock in the second right premolar tooth. The attacks were more frequent than before

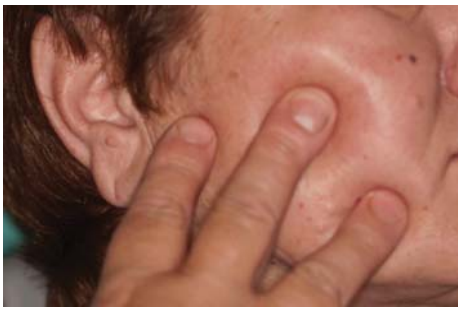

Figure 1: The patient showing the painful area.

Table 1: The patient's results of laboratory tests (with reference values [28]).

Various blood laboratory tests	Laboratory test results	Reference values
Blood glucose levels before eating	9.6 mmol/l	for DM <7 mmol/l
Blood glucose after eating	18.3 mmol/l	<11.1 mmol/l
Cholesterol	5.9 mmol/l	<5 mmol/l
LDL cholesterol	3.3 mmol/l	<3 mmol/l
HDL cholesterol	1.7 mmol/l	<1.2 mmol/l for women
Triglycerides	1.95 mmol/l	<1.7 mmol/l
Glycosylated hemoglobin HbA1c	59 mmol/mol	<42 mmol/mol

Table 2: An abnormal blink reflex finding induced by the stimulation of the supraorbital nerve on the right side (with reference values [17,18]).

Blink reflex components	Blink reflex finding	Reference values
Early response R1	13.2 ms	> 12 ms
Late (homolateral) R2 response	45.4 ms	> 42.4 ms
Contralateral R2c response	45.2 ms	> 44.4 ms
Ipsilateral R3 response	122 ms	> 0 ms*

* Every appearance is nociceptive.

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and after root canal treatment of the second right premolar and the taking of antibiotics (amoxicillin with clavulanic acid). The radiologic examination excluded odontogenic etiology. Also, the electric shock sensation spread diagonally from the second right premolar tooth towards the ear and the patient felt a burning sensation in the mouth and numbness of the right side of the tongue. In that period, the pain appeared in the morning when she got up and it ceased around 2 pm, and then returned in the evening during rest, for example, when she was watching television. The problems caused limited mouth opening so that she could not eat apples and other food harder to chew, and often she could not drink coffee from a cup or speak. The patient recollected losing at least one or two teeth distally from tooth 44 with similar symptomatology. Prior to neurologic treatment, she visited an oral medicine specialist.

Dental and functional examination

The patient wore an upper partial acrylic denture and crowns on the anterior teeth, which were two years old. The supporting zone of the teeth in the lower right segment was not prosthodontically replaced. The patient had suspected gingivitis and visibly poor oral hygiene, but with a preserved alveolar bone margin which was visible on the panoramic X-ray image. Condyles were symmetrical, and the right styloid process elongated, but the patient denied feeling any pain on swallowing.

Targeted diagnostics excluded symptomatology related to temporomandibular disorders (TMD)

Diagnostic Criteria (DC) for TMD [31] were used as well as manual examination techniques (manual functional analysis) according to Bumann and Groot Landeweer [32]. The clinical examination did not show any pathology related to masticatory muscles and TMJs, there was neither pathologic noise in TMJs, nor any pain on mouth opening (active mouth opening amounts to 48 mm now, the patient states that in the period of symptoms' manifestation, it amounted to 33 mm or less). On palpation, there is neither pronounced anterior condyle movement, nor any subluxation settling across the tuberculum dextrally. Passive compressions did not provoke pain in the TMJ. Magnetic resonance imaging ("Harmony", Siemens, Erlangen, Germany) was performed by weighted (TR 450/TE 12; matrix 256 x 192; 160 x 160 field of view) and T2 weighted images (TR 3000/TE 66; matrix 389 x 512; 190 x 190 field of view) showed physiologic relations in both TMJs with the hyperextension of both condyles about 1 cm across the zenith of the tuberculum. Both in open and closed mouth positions, the discs were in physiologic relation with the condyles and without any signs of osteoarthritis or effusion of articular spaces (Figure 4).

Psychological measurement

From the patient's history and medical records, it was clear that due to complex painful symptoms, the patient took drugs related to her mental condition. She mentioned that several times, during the TN attacks, she experienced additional stressful events, although she also took several drugs due to her diabetic polyneuropathy: benzodiazepine anxiolytic alprazolam (Helex, 0.5 mg, 1 pill/day); tricyclic antidepressant

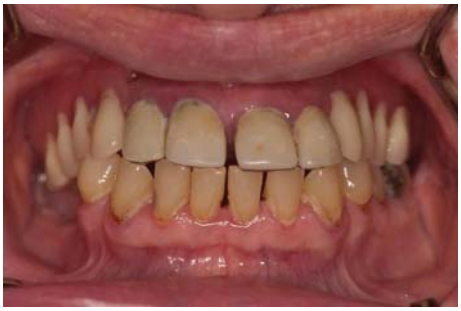

Figure 2: Intraoral view of the patient.

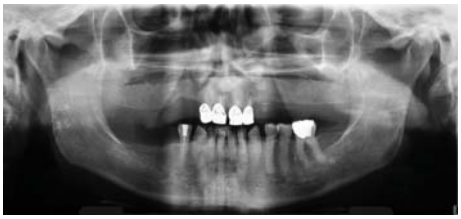

Figure 3: Panoramic radiographs of the patient.

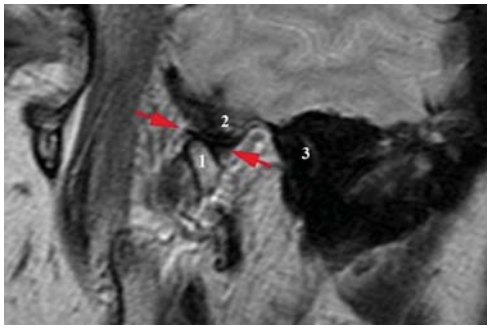

Figure 4: Temporomandibular joint on the parasagittal plane on T2 weighted MR image with hyperextension condyle position: 1 - Condyle head, 2 - Articular eminence, 3 - External auditory meatus, and arrows - physiological disc position.

amitriptyline (Amyzol 25 mg, 2 pills 3 hours before bedtime), as well as an opioid analgesic tramadol (Tramal, 3 x 10 drops, 10 drops = 25 mg).

The psychological assessment was carried out by Spielberger's State-Trait Anxiety Inventory (STAI) Form Y [33]. STAI is a commonly used measure of self-reporting anxiety on a four-point Likert scale. The range of scores is 20-80, with the higher score indicating greater anxiety. STAI 1 test measures anxiety as a subjective state, a feeling lasting for a week, including the day of testing, and STAI test 2 measures anxiety as a relatively stable individual characteristic during life in general. According to Spielberger for the group of subjects aged 50≥, borderline values for the female gender are: 32.20 for STAI 1 and 31.79 for STAI 2. The patient showed high levels of anxiety on both scales (STAI 1: 46, STAI 2: 58).

Three-years follow-up

After the neurologic and dental treatment, the patient continued the therapy for her basic illness (DM type 2), hyperlipidemia, while the neuralgia was treated with vitamin B only. Mouth opening was painless, amounting to 46 mm,

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but she still complained about the burning sensation, for example, when eating an apple, which appears in the mandible dextrally and under tooth 44. She also had numbing pain along the zygomatic bone on the right side of the face and towards the right eye. In conclusion, although the severe attack did not recur, the patient's subjective opinion is that there was no improvement in her condition. The result of the laboratory test for HbA1c was 59 mmol/mol (<42 reference value), which reveals insufficient control of the hyperglycemia, that is, of DM.

Discussion with a literature review

In general, DM is a public health problem, since its prevalence in populations older than 20 is expected to rise from 451 (in 2017) million to 693 million by 2045. DM treatment (that is, the treatment of hyperglycemia) is challenging because it often includes accompanying diseases: obesity (80%), hypertension (60%), and dyslipidemia (30% - 40%) [5].

It is a diagnostic challenge to differentiate nociceptive (somatic) and neuropathic pain and, subsequently, treat it correctly [3]. TMDs are characterized by physiologic activation of nociceptors as a response to musculoskeletal tissue damage (for example, TMJs), whereas neuropathic pain is a lesion of the somatosensory nervous system, thus causing abnormal activity of the nociceptive pathways [1,2]. In the pathophysiology of the genesis of the painful symptoms, both peripheral and central mechanisms have been proposed to play an important role. Damage to peripheral nerves results in hyperexcitability in primary afferent nociceptors (peripheral sensitization) which in turn leads to hyperexcitability in central neurons (central sensitization) and generation of spontaneous impulses within the axon as well as within the dorsal root ganglion of these peripheral nerves [9,10].

According to Alajbegovic, et al. [11] DM duration is the key factor for the development of microvascular complications of neuropathy. In our study, the risk was greater in female patients with type 2 diabetes. The prevalence of diabetic polyneuropathy is considered to be up to 50.8% of patients with DM type 2, depending on the design and sample of the study. 32.8% of patients with diabetic polyneuropathy had neuropathic pain, which was a prevalence of 17.9% for patients with DM type 2. Only 50% of patients with diabetes used analgesic pharmacotherapy, out of which 28% is under recommended treatment for neuropathic pain (anticonvulsants or antidepressants). The opiate derivative tramadol is helpful in the management of painful diabetic peripheral neuropathy, regardless of its risks of addiction [15].

In most cases, cranial nerve involvement in diabetics (range 3% - 14%) relates to motor neuropathies, causing acute onset of ophthalmoplegia. Multiple cranial nerve neuropathies can occur simultaneously in the diabetic population but they can also be the first clinical signs of recently diagnosed diabetes. A clinical manifestation of cranial nerve neuropathies is palsy of involved cranial nerves with an incidence of 0.19% in diabetic patients compared with an incidence of 0.13% in the non-diabetic population [21].

There is a possible relation to poly cranial neuritis characterized by damage to sensory and motor nerves which has been found in diabetics with Ramsay Hunt syndrome, which manifests as a sensory and motor complication of herpes zoster infection, including the trigeminal nerve [34]. Tu, et al. [21] presented a clinical case of multiple cranial neuropathies with four episodes of oculomotor and facial nerve palsy, involving the left oculomotor, trochlear nerve, and possible ophthalmic division of trigeminal nerve.

Trigeminal nerve appears to be rarely involved in patients with diabetes mellitus. Cruccu, et al. [23], Urban, et al. [24], Wong, et al. [25], and Takayama, et al. [26] are examples of very few existing reports of the relationship between diabetes and TN- related orofacial pain. Cruccu, et al. [23] found sensory trigeminal neuropathy in 8 out of 15 diabetic patients with chronic inflammatory demyelinating polyneuropathy, which causes subclinical neuropathy to its mandibular branch. Urban, et al. [24] researched trigeminal and facial nerve functions electrophysiologically and concluded that their involvement in diabetes-related polyneuropathy can be expected, although limb nerve involvement is significantly more frequent.

Wong, et al. [25] described the involvement of the oculomotor nerve and the maxillary division of the trigeminal nerve in a case of a 53-year-old man who suffered from pain and swelling in the right infraorbital region in the area of the right maxillary canine. He had poorly controlled insulin-dependent diabetes mellitus complicated by peripheral neuropathy, hypertension, and migraine headaches. Contrary to the satisfactory CT findings of the patient described in our paper, in the above-mentioned case, the CT showed generalized atrophy and a focal region of low attenuation in the left frontal lobe and central pons. The left frontal lobe attenuation was consistent with encephalomalacia and likely represented a previous infarction. Takayama, et al. [26] described bilateral trigeminal nerve involvement in a 69-year-old male patient with DM type 2. Neuropathy of ophthalmic and maxillary branches was characterized by causalgia and dysaesthesia in the patient's cheeks and around his eyes. Attack episodes were related to the level of successful glycemic control.

Patients with chronic inflammatory demyelinating polyneuropathy were a sample for research in the study of Kokubun and Hirata [35], and using neurophysiological tests, they found that subclinical trigeminal and facial neuropathies were extremely high (60% - 85%).

The best indicator of metabolic control is glycosylated hemoglobin (HbA1c), which is a fraction of hemoglobin to which the glucose from the blood bonds independently of insulin; hence, it is directly dependent on the average level of glucose in blood [28]. HbA1c value shows the success of diabetes control in the previous two to three months. The laboratory test results (HbA1c) of the reported case of the female patient did not show successful control of DM type 2. However, Takayama, et al. [26] noted that symptoms had become more severe when HbA1c increased to as high as 89 mmol/mol. When the patient took insulin therapy, the level of HbA1c decreased to 51 mmol/mol. They concluded that better glycaemic control improved the patient's clinical picture related to neuropathic pain.

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The finding of the blink reflex test of this patient supports the diagnosis of trigeminal neuropathy, which shows irritative changes to the afferent branch of the reflex arc dextrally [36]. Mikula, et al. [37] used the blink reflex method and concluded that it can be useful in differential diagnostics: the BR may prove a significant aid in distinguishing the idiopathic TN (normal latencies R2 and R2c) and symptomatic (prolonged latencies R2 and R2c) disease types. The incidence of the R3 component was higher (84%) in patients with idiopathic TN than in patients who suffered from symptomatic TN (20%).

The biopsychosocial conceptualization of the pain experience recognized psychological factors as a part of the multidimensional description of pain; especially chronic pain conditions such as diabetic polyneuropathy. Since DM type 2 is a chronic disease, apart from timely diagnosing, regular controls with treatment modifications if necessary, a psychosocial factor of facing the patient with the disease and accepting a healthy lifestyle is very important [15]. Anxiety is the most common affective disorder and a great problem for patients with DM type 2 because as many as 19.8% of them have a diagnosis of anxiety disorders [38]. In DM treatment, psychological evaluation is necessary because psychological management improves quality of life, and has positive impacts, particularly on painful diabetic polyneuropathy. Successful treatment of idiopathic TN is challenging. There is no gold standard for assessing neuropathic pain caused by idiopathic TN. In order to successfully treat this type of orofacial pain, which can be characterized as severe pain without a clear etiopathogenesis, a collaboration between the dentist and the neurologist and active participation from the patient's side are needed [39]. Although the dentist is not the primary health care provider who decides on the choice of pharmaceutical therapy, the intensity of symptoms in the stomatognathic system should be taken into account, in particular the involvement of toothache and intensity of neuralgic attacks without a dental cause.

Conclusion

Sensory complaints in the area of the teeth, jaws, and mouth often escape notice or remain undiagnosed. A multidisciplinary approach is needed for a proper understanding of the causes as well as for treatment planning in cases of possible co-morbidity with orofacial neuropathic and/or nociceptive pain. Beyond the scope of dental medicine, and biochemical diagnostics, HbA1c has helped in the interpretation of both therapeutic efficacy and the risk of persistent diabetic complications, such as in cases of neuralgiform pain of the orofacial region. In addition, the blink reflex method can be a useful diagnostic tool to distinguish asymptomatic patients from symptomatic patients with TN.

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