Diabetic thoracic polyradiculopathy: a forgotten pain

Polirradiculopatía torácica diabética: un dolor olvidado

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Abstract

The diabetic thoracic polyradiculopathy (DTP) is an uncommon neuropathic manifestation that usually appears in patients with long evolution diabetes together with microangiopathic chronic complications. It appears as a painful picture that affects the thoracic and abdominal region depending from the affected roots. The pain can be of great intensity and disabling and very often the diagnosis is delayed when assessing other more frequent causes first. The diagnosis is confirmed through an electromyography, having to rule out other organic etiologies, specially the tumoral ones. The treatment includes the most used drugs for painful diabetic neuropathy, being sometimes the administration of corticosteroids, transcutaneous neural stimulation or plasmapheresis required. We present the case of DTP in a young patient with a recent diagnosed T2D with an undetermined evolution time and with serious microangiopathic manifestations, standing out the characteristics of the medical profile, the diagnostic approach and the therapeutic attitude.

Keywords: diabetes mellitus, thoracic polyradiculopathy, abdominal pain, electromyography.

Case report

Patient aged 40, native of Madrid, without pathologies of interest or toxic habits, who attends due to hyperglycemia. Family history: mother diagnosed with T2D treated with insulin. He attends since he presents symptoms of a 4-months evolution progressive weakness of the lower limbs (LL), with distal paresthesia, loss of 8 kg of weight, blurred vision, nycturia of 1-2 times and difficulty to keep satisfactory sexual relations. Due to the vision alterations, he attends the ophthalmology service, where the presence of proliferative diabetic retinopathy is detected.

The physical exploration points out: height 167.5 cm; weight 63 kg; body mass index 22.4 kg/m²; blood pressure 190/100 mmHg. Thyroids level 0. Heart auscultation: tachycardia to 110 heartbeats/minute with systolic ejection murmurs of functional aspect. Soft and depressible abdomen, without pain and without visible ecchymosis. White stretch marks due to distension. LL without edemas, conserved peripheral pulses. Reduced osteotendinous reflexes. Reduced vibratory sensitivity. Sensitivity with conserved monofilament.

Complementary data: red blood cells 3.93 × 10⁶/mm³; Hb 11.1 g/dL; hematocrits 32.3%; MCV 82.2 fL; platelets 291 × 10⁹/mm³; leukocytes 6.45 × 10⁹/mm³ (60% S, 26% L, 14% M);
ESR 94 mm/h; CRP 0.6 mg/dL; glucose 488 mg/dL; total cholesterol 319 mg/dL; cHDL 68 mg/dL, cLDL 211 mg/dL; triglycerides 198 mg/dL; urea 52 mg/dL; creatinine 1.8 mg/dL; creatinine clearance 38 mL/min; GOT 12 U/L; GPT 13 U/L; GGT 55 U/L; LDH 360 U/L; FA 39 U/L; CK 80 U/L; Ca 9.6 mg/dL; P 5.0 mg/dL; Mg 2.37 mg/dL; ferritin 640 ng/mL; total proteins 7.0 g/L; albumin 51.6%; alpha-1-globulin 4.7%; alpha-2-globulin 14.8%; beta-2-globulin 14.4%; gamma globulin 14.5%; CEA 3.41 ng/mL; alpha-fetoprotein 5.12 ng/mL. Urine: density 1,010 g/mL; pH 7.0; proteins 150 mg/dL; glucose 1,000 mg/dL; negative ketone bodies; erythrocytes 20/µL; sediment with 8-15 erythrocytes/field. Basal cortisol 15.9 µg/dL; cortisol in urine 62 µg/24 h; albumin in urine 6,418 mg/24 h. TSH 2.01 mU/mL. Total testosterone 5.6 ng/mL. HbA1c 13.7%. Basal C-peptide 2.06 ng/dL; Anti-GAD antibodies and negative anti-IA2 antibody. X-ray of thorax: without findings. Electromyogram (EMG) of LL: compatible with mixed sensitive-motor polyneuropathy (axonal and demelanizing) and of proximal predominance, of moderate intensity in both LL and mild in higher limbs. Thoracoabdominal without contrast: absence of findings related to the painful sensation. A marked gastric distension can be observed and a very distended urinary bladder with thickened walls diffusely. Multiple and important calcifications in the vas deferens, the cavernous bodies and the aorto-iliac axis.

Treatment is started with a diabetes diet of 2,200 kcal, insulin glargine 14 U/day, olmesartan 40 mg/day; AAS 100 mg/day, atorvastatin 40 mg/day, vitamin B complex and simple analgesics with the diagnosis of T2D, proliferative retinopathy, diabetic nephropathy with nephrotic syndrome and renal failure stage III, diabetic neuropathy with sensitive-motor neuropathy of lower limbs and erectile dysfunction, obtaining a fast and evident clinical recovery parallel to the metabolic control. The initial dose of insulin does not require practically later adjustments considering the glycemic profiles. The HbA1c after 2 and 5 months after his first visit is of 7.7 and 6.3% respectively.

In spite of this initial improvement, a month later the patient starts showing a very intense abdominal pain, which is constant and localized in the top middle part of the abdomen. Then, a burning feeling at interscapular and thoracic level is added. The patient as an adverse effect of the insulin initially relates the pain. Therefore it is decided the change to insulin detemir in spite of not showing local signs of secondary irritation to the injections. However, the pain increases, contrasting with the absence of abdominal exploratory findings of interest. Thus, an EMG is performed of the abdominal rectus muscle and an electroneurogram (ENG) of the right intercostal nerve VIII with electric stimulation of the costal ridge in the anterior rectus muscle of the abdomen. The results determine an EMG with a chronic neurogenic pattern: increase in the duration of the motor unit potential and of the incidence of polyphasia (figure 1). The ENG shows normal values of motor conduction and a reduction of amplitude of the motor evoked potential; these findings are compatible with a radiculopathy of right T8 root (figure 2). The neurogenic affection of the muscle is attributable to a radicular affection as the conduction speed of the nerve is normal and the reduction of the amplitude of the response reflects a reduction in the density of the nervous fibers.

In spite of the findings compatible to the DTP, an abdominal TC is performed in order to rule out other causes of abdominal pain, without encountering related findings, though indeed an important gastric and gallbladder dissention compatible to a neuropathy in the autonomous system at such levels.

Treatment is started addressed to the neuropathic pain, with pregabalin 75 mg/8 h, and a satisfactory evident response is obtained. However, side effects appear (dizziness and edemas) that oblige to the reduction of the dose (75 mg/12 h), adding then duloxetine 60 mg/day, with a clear clinical improvement and a good tolerance. The initial pain disappeared with such combined treatment, persisting only some mild paresthesias and dyesthesias in the interscapular region.
Comment

The commented case report is placed in the context of a recent diagnosed diabetes. Factors as age, the absence of obesity and the relevant loss of weight led to think, initially, that it was a T1D. However, other data, as the absence of ketosis, the relevant presence of C-peptide, the negativity of pancreatic auto-immunity and especially the presence of important micro-angiopatia complications led to the diagnosis of T2D of undetermined evolution time.

Though the presence of chronic complications related to T2D at the moment of the diagnosis is not infrequent; the presence of multiple and advanced micro-angiopatia complications is eye-catching in a young adult without known pathological history.

The seriousness of the nephropathy might led us to think in other etiologies different from the diabetes considering the high globular sedimentation rate (GSR) and the presence of a mild hematuria in the initial sedimentation, though this last one was not confirmed in later analysis. However, the GSR is high in almost all the patients with nephrotic syndrome or advanced renal failure, two-thirds of which show values of >60 mm/h\(^2\) this is attributed to plasmatic factors, in particular at high levels of fibrinogen, existing a direct relation between the level of proteinuria and the GSR.\(^3\) It has to be pointed out that both the renal and ophthalmologic affection showed a very aggressive course during the short follow-up of the patient (8 months), who required retinal panphotocoagulation and vitrectomy, and at present the patient is in situation of predialysis. Therefore, the study of the gastric and gallbladder affection observed in the imaging tests and that do not cause relevant manifestations has been postponed at the request of the patient himself.

The abdominal pain is a symptom subject to a wide differential diagnosis, within which a group named “pain of the abdominal wall” can be observed.\(^1\) One of the causing pathologies of this pain of the abdominal wall is the T7-T12 radiculopathy, whose most outstanding etiologies are the diabetes and the herpes zoster.

The DTP is an infrequent cause of neuropathic affection in the diabetic patient and consists of the affection of the nerve roots of the thoracic spine. This causes a predominantly sensitive alteration, though also motor and autonomic, of the nervous plexus that depend from the involved roots.

The DTP might appear in patients with long evolution diabetes along with other chronic complications, though isolated recent onset diabetes cases have been published.\(^4\)

Since its infrequency, it is not usually suspected on the existence of DTP regarding to a diabetic patient with abdominal pain. Precisely and taking into account the abdominal reassociation of DTP concerning to a diabetic patient with abdominal pain. Antidepressants, antiepileptics, transcutaneous neural stimulation, corticoids, intravenous immunoglobulin and plasmapheresis have been used, and it has been frequently required the use of opioids due to the intense referred pain.\(^5\)\(^6\)\(^7\)\(^8\)\(^9\)\(^10\) In our case, the therapeutic response to the pregabalin in combination with duloxetine was satisfactory and the patient was relieved from an incapacitating pain due to its intensity and chronicity.

To conclude, we want to point out the importance of taking into account this entity when we are facing a diabetic patient with abdominal pain that cannot be explained by other causes, which will aim to the etiological differentiation, will reduce the diagnostic tests and will anticipate the adequate treatment.

Declaration of potential conflict of interests

A. Arranz, L. Nattero, P. Rojo, B. Santana, A. Azcárate, I. Tejado and A. Gómez-Pan state that there are no conflicts of interest as regards to the content of this article.

References