CASE REPORT

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Painless Footdrop in a Child with Newly Diagnosed Type 1 Diabetes Mellitus: Case Report

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What is already known on this topic?

Diabetic neuropathy is a major cause of morbidity amongst diabetics. Its presentation is usually regarded as a late-stage complication of diabetes, mostly affecting patients with advancing age

What this study adds?

This report describes a rare case of a pediatric patient with newly diagnosed type 1 diabetes mellitus (T1DM) who presented with signs of mononeuropathy. It highlights that T1DM may present atypically as acute onset neuropathy in pediatric patients, making it an important differential diagnosis.

Abstract

Diabetic neuropathy is a major cause of morbidity among diabetics, usually affecting patients with long-standing diabetes and advancing age. We present a case of atypical first clinical presentation of type 1 diabetes mellitus (T1DM) in a pediatric patient. A 15-year-old male patient presented to the emergency department with complaints of right foot weakness associated with mild paresthesia of 1-week duration. There were complaints of polyuria, polydipsia and weight loss in the same timeframe. On subsequent examination, the patient exhibited signs of right-sided foot drop with weak ankle dorsiflexion and eversion, accompanied by impaired sensation over the dorsum of the right foot. Lab results confirmed a diagnosis of T1DM and the patient was started on subcutaneous insulin injections. The patient's foot drop recovered within one month of insulin initiation. This case highlights that T1DM may present atypically as acute onset neuropathy in pediatric patients, making it an important differential diagnosis.

Keywords: Case report, diabetes mellitus, footdrop, mononeuropathy

Introduction

Diabetic neuropathy (DN) is a major cause of morbidity amongst diabetics (1). It is a well-known process that over half the individuals with diabetes develop over time (1). Its presentation is usually regarded as a late-stage complication of diabetes, mostly affecting patients with advancing age (1). In pediatric patients, DN is rarely seen, especially as an initial presentation of type 1 diabetes mellitus (T1DM). This paper describes a rare case of a pediatric patient with newly diagnosed T1DM who presented with Foot drop.

Case Report

A previously healthy 15-year-old Arab male presented to our hospital with the chief complaint of right foot weakness of 1-week duration. The weakness was associated with a mild tingling sensation in his right foot but no numbness.

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The patient also reported feeling unbalanced, often tripping over, as well as dragging his right foot for the past week. During this period, symptoms of polydipsia, polyuria, and polyphagia were also present.

Upon detailed history questioning, the patient reported right knee trauma two months prior to this presentation. He described two days of pain and clicking sounds in the affected knee, which resolved spontaneously and has been asymptomatic since. No cast was worn. No recent infections were reported. Past medical history and family history were unremarkable.The patient did not take any regular medications.

The patient was seen by the neurology team in view of his presenting symptoms. The neurological examination revealed a high steppage gait with right-sided foot drop and absent heel strike. Mild wasting of calf muscles was noted in the right leg. Examination of the right foot showed a grade 0/5 ankle dorsiflexion and 0/5 ankle eversion with normal ankle plantar flexion and normal ankle inversion. Ankle reflex was absent on the right and superficial sensation was also impaired over the dorsum of the right foot. The remainder of the general physical and neurological exam findings were normal, including left foot, upper limbs, cerebellar and cranial nerve functions.

Nerve conduction studies (NCS) were performed twice on bilateral peroneal, tibial, sural and superficial peroneal nerves (Tables 1, 2, 3). The tests showed normal distal latencies, conduction velocities and amplitudes from all of the nerves tested.

The diagnosis of T1DM was made after the laboratory reported a random serum glucose level of 247 mg/dL (normal 73-112 mg/dL) with hemoglobin A1c (HbA1c) of 11.7% (normal 4.3-5.7%) and anti-glutamic acid decarboxylase antibody titers of 51.9 units/mL (normal <5 units/mL).

Table 1. Motor nerve conduction study findings. Peroneal nerve responses to the extensor digitorum brevis muscle and tibial nerve responses to the abductor hallucis muscle were symmetrical with normal responses and amplitude, bilaterally

Site	Lat. (ms)	Dur. (ms)	Amp. (mV)	Area (mVms)	Stim. (mA)	Dist. (mm)	Intvl. (ms)	NCV (m/s)
Left peroneal								
Ankle	4.3	12.5	8.5	26.5	25		4.3	
Head of fibula	11.6	16.7	7.5	26	37	320	7.4	43.5
Right peroneal								
Ankle	4.1	12.3	8.9	23.9	21		4.1	
Head of fibula	11.5	12.2	7.6	20.9	32	340	7.4	45.9
Left tibial								
Ankle	3.7	14.2	21.4	81.9	21		3.7	
Popliteal	13.3	16	14.5	71.8	50	400	9.6	41.7
Right tibial								
Ankle	4.3	12.1	22.3	77.8	20		4.3	
Popliteal	13.5	13	15.5	64.8	41	380	9.2	41.3

Table 2. Sensory nerve conduction study results. Sensory responses from the superficial peroneal and sural nerves bilaterally showed normal amplitudes

Nerve	Lat. 1 (ms)	Lat. 2 (ms)	Amp. (uV)	Area (mVms)	Stim. (mA)	Dist. (mm)	Intvl. (ms)	NCV (m/s)
Left sural	2.4	3.1	33.8	2.1	23	110	2.4	45.8
Right sural	2.9	3.5	40.2	2	18	120	2.9	42.1
Left superficial peroneal	2.2	2.7	23.6	0.8	22	110		43.5
Right superficial peroneal	3.1	2.2	14.3	1.5	24	120		45

Table 3. F-wave latency results. Bilateral peroneal and tibial F-wave latencies were within normal limits

Nerve	Side	Stim. Site	F-Lat.	F-M Lat.
Peroneal	Left	Ankle	46.4 ms	42.2 ms
Peroneal	Right	Ankle	46.9 ms	46.9 ms
Tibial	Ankle	Ankle	49.2 ms	45.9 ms
Tibial	Ankle	Ankle	46.9 ms	43.5 ms

Venous blood gas showed normal pH and bicarbonate values. Moderate glycosuria and ketonuria were noted on urinalysis. Other laboratory findings, including thyroid functions, infectious workup, electrolytes, celiac screen and full blood counts were within normal ranges. Trauma and masses as a cause of peroneal neuropathy were ruled out by normal findings on magnetic resonance imaging (MRI) of the right knee. Central causes, including space-occupying lesions, infarction, bleeding, and inflammation processes were all ruled out by normal MRI brain and spine findings.

The patient was admitted and started on long acting insulin Degludec once daily and short acting insulin Aspart with meals at a total daily dose of 1.2 units/kg/day. His insulin doses were adjusted based on his blood sugar readings during hospital admission. He also underwent a few sessions of physiotherapy during his admission.

Within three days his glucose levels had normalized and he had mild improvement of his right foot weakness. The patient was discharged four days after his admission with a plan to follow up in physiotherapy, endocrinology and neurology outpatient clinics. Both the patient and his parents were given comprehensive education about diabetes and plans for follow up.

Two months later, in the follow up endocrinology clinic visits, the patient's glycemic control had significantly improved (HbA1c 6.4%) and he was asymptomatic.

Discussion

DM is one of the most commonly diagnosed endocrine disorders among children (2). T1DM is characterized by elevated levels of blood glucose as a result of autoimmune destruction of pancreatic beta cells, which cause insufficient insulin production (3). There has been a steady increase in the incidence of DM worldwide, with T1DM rising 3% annually in the last few decades (4). The United Arab Emirates and the Middle East in particular are facing an epidemic, with the Middle East and North Africa region currently exhibiting the highest prevalence of diabetes in the world after age-standardization (5).

The first presentation of DM usually involves osmotic symptoms, such as polyuria, polydipsia and weight loss (6). If left undiagnosed for long, patients may also present with diabetic ketoacidosis (DKA), which poses significant risks to the patient's morbidity and may even be fatal. This makes the early diagnosis of diabetes and recognition of atypical symptoms very important (7).

DN is a widely known complication of long-standing diabetes (1). Multiple mechanisms have been suggested to play a role in the pathogenesis of DN. These include hyperglycemia-induced oxidative damage, nerve ischemia due to endothelial dysfunction and the loss of insulin and its role as a neurotropic peptide (8,9,10).

DN can be divided into two broad categories. The first is generalized neuropathy, which encompasses diabetic sensorimotor polyneuropathy. This group has a chronic presentation that typically correlates with longstanding diabetes and advancing age (11). The second group encompasses a more acute presentation, has a self-limiting course, and is not associated with duration, intensity of diabetes or hyperglycemia. Entities within the second group include painful sensory neuropathy with weight loss (or diabetic cachexia), treatment related (insulin neuritis), polyneuropathy after ketoacidosis and hyperglycemia neuropathy (12).

The present case report appears appropriate for the second group, as it was acute in nature and had a rapid resolution following treatment. The presented patient had a predominant motor involvement, and no reports of weight loss, which makes diabetic cachexia less likely as it is mainly a sensory neuropathy (12).

Baszyńska-Wilk et al. (13) described a 9-year-old patient who developed symmetric lower limbs paresis with new onset T1DM after being admitted with severe DKA. During the course of her stay, the authors reported findings of brain edema and multifocal vasogenic brain lesions on further imaging. Their case exhibited a clinical feature of peripheral neuropathy after DKA, a complication that can be a consequence of peripheral nerve ischemia or other hemodynamic and metabolic changes that are linked to DKA (12). In our case, this diagnosis was taken into consideration, however our patient had no laboratory results suggestive of DKA and also showed no signs of central nervous system abnormalities, which are expected in DKA.

Multiple non-diabetic etiologies were also considered. Peroneal nerve injury as a result of trauma was investigated. However, the time frame of the patients' knee injury in relation to the onset of the symptoms made this very unlikely. Furthermore, our patients' symptoms coincided with the classic presenting symptoms of T1DM and rapidly improved with insulin treatment, all pointing to DN as the cause of his symptoms.

Few cases of pediatric neuropathy in the setting of undiagnosed diabetes have been observed and are accessible in the literature (14,15,16). Our case closely resembles the aforementioned cases in the signs and symptoms of our patients, aside from a normal NCS result. Although abnormal NCS results may be frequently found in

neuropathies, it has been reported to require a longer course of diabetes and a higher severity of hyperglycemia (17). In addition, the presented patient's neuropathic symptoms also completely recovered following glycemic control. This reinforces our diagnosis of DN as an atypical presentation of diabetes.

Conclusion

This case highlights that T1DM may present atypically as acute onset neuropathy in pediatric patients. It is important to recognize these clinical features as early recognition can reduce the risk of further complications and allows patients to receive appropriate treatment.

Ethics

Informed Consent: Written informed consent was obtained from the patient's guardian.

Authorship Contributions

Surgical and Medical Practices: Manal Mustafa, Samar Almuntaser, Concept: Maryam Jafari, Samar Almuntaser, Design: Maryam Jafari, Ahmedyar Hasan, Jessie Joseph, Data Collection or Processing: Maryam Jafari, Manal Mustafa, Samar Almuntaser, Analysis or Interpretation: Maryam Jafari, Samar Almuntaser, Literature Search: Maryam Jafari, Ahmedyar Hasan, Jessie Joseph, Samar Almuntaser, Writing: Maryam Jafari, Ahmedyar Hasan, Jessie Joseph, Manal Mustafa, Samar Almuntaser.

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