



# Acute mononeuropathy in an 8-year-old girl with newly diagnosed type 1 diabetes



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Disclosure Statement: Authors declare that they have no conflict of interest.

## Introduction and purpose

- Diabetic neuropathy (DN) consists of a variety of clinical entities including even acute mononeuropathies. Their pathogenesis has not been elucidated. The most commonly affected region, sciatic nerve (SN), was recently proposed to suffer the most severe molecular consequences of diabetes in the nervous system.
- Our purpose is to present an 8-year-old girl diagnosed with acute mononeuropathy of SN during the course of severe diabetic ketoacidosis (DKA).

## Case report

### History

- An 8-year-old girl was admitted to the Emergency Department because of *Kussmaul breathing* during the last half hour, accompanied by *polydipsia*, *polyuria* and fatigue over the last 48 hours.
- Her personal history was uneventful except for recurrent wheezing, while her family history was not significant.

### Physical and Laboratory Evaluation

- On admission, she was conscious, space and time-orientated. Physical examination revealed body temperature of 36.5°C, blood pressure of 119/72 mmHg, pulse of 164 beats/minute and oxygen saturation of 98%, *acetone breath*, signs of *severe dehydration*, impaired capillary refill time and mild generalized abdominal tenderness.
- She was *acidotic* (pH 7.09, HCO<sub>3</sub><sup>-</sup> 1.7 mmol/l) with initial random *glucose* of 25.8 mmol/l. Type 1 diabetes (T1D) was diagnosed based on hyperglycaemia and severe ketoacidosis and confirmed by elevated glycated haemoglobin (HbA1c), diminished c-peptide and positive glutamic acid decarboxylase autoantibodies.

### Course

- The patient was managed with *aggressive fluid resuscitation* and *continuous intravenous insulin infusion*. Because of lethargy and acidosis deterioration, she was transferred to *pediatric intensive care unit*, where she made a gradual and uneventful recovery and was discharged to our department after five days for education by a specialized pediatric diabetologist.
- During the second day of her readmission, *right foot drop* was observed (Figures 1,2), accompanied by regional redness and mild edema, producing a steppage gait.

Figure 1. Right foot drop



Figure 2. Right foot drop



## Investigation

- Ultrasonic triplex scanning of lower extremity arteries and veins, magnetic resonance imaging (MRI) of brain and spine and bone scintigraphy revealed normal findings and osteomyelitis and thrombophlebitis were excluded.
- Electrophysiological study revealed reduced nerve conduction velocities (NCV) indicative of severe axonal damage of right SN (Table 1).

Table 1. Results of electrophysiological study

	Tibial nerve (right / left)		Peroneal nerve (right / left)	
Motor amplitude	-	16.14 mV	-	2.76 mV
Sensory amplitude	1.8 µV	7 µV	8.10 µV	15.10 µV

- An infectious etiology was not identified. Cyanocobalamin, folic acid, thyroid stimulating hormone and free T4 levels were within normal ranges.

## Diagnosis

- Acute mononeuropathy of SN during the course of severe DKA

## Treatment

- She started physiotherapy and was treated with B6 and B12 vitamins and magnesium without clinical or electrophysiological improvement eight months later.

## Follow-up

- Four months later, although she has an optimal glycaemic control, no clinical or electrophysiological improvement was recorded. She continues physiotherapy.

## Conclusion

- DN is not only its chronic and generalized variant described merely in adults. It is a heterogeneous group of entities also including acute and focal neuropathies affecting rarely children even in the course of DKA. SN was recently proposed to suffer the most severe molecular consequences of diabetes in the nervous system. Pediatricians should be aware of such potential when interfering with cases of DKA. It would be of great pathogenetic and therapeutic interest to search for molecular consequences of DKA in SN, as they may start to develop too early.

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