ACUTE PAINFUL NEUROPATHY IN A TEENAGER WITH TYPE 1 DIABETES (T1D) AND EATING DISORDERS

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Background

Acute painful neuropathy (APN) is a rare manifestation of diabetic neuropathy (DN) in adolescents with type 1 diabetes (T1D), which has been associated with poor metabolic control, as depicted by elevated levels of glycosylated hemoglobin A1c (HbA1c). Eating disorders, such as binge eating, bulimia, anorexia nervosa and other disorders not otherwise specified, have been described in T1D adolescents and have been linked with metabolic derangement.

Case presentation

A 12 year old teenage girl complained of persistent pain and numbness in upper and lower limbs. She was diagnosed with T1D 9 months ago and received intensive insulin treatment. During the last month she reduced food intake and omitted insulin doses resulting in diabetic ketoacidosis. She presented a 2kg weight loss [BMI reduction: 14.1 kg/m^2 (10th centile) →13.2 kg/m^2 (3rd centile)] during 3 months and her metabolic control deteriorated progressively (HbA1c: 11%). After the management of diabetic ketoacidosis, the neurologic symptoms persisted. The detailed neurological clinical examination revealed normal tendon reflexes but elevated vibration sensation thresholds. Nerve conduction studies (NCS) exhibited abnormal values in the peroneal nerve [sensory action potential (SNAP): 2.7mV, sensory conduction velocity (SCV): 35msec] and normal sural nerve measurements (SNAP: 10uV, SCV: 50msec). Assessment of autonomic neuropathy with pupillometry revealed no abnormality. Psychologic and psychiatric support was immediately initiated concerning her eating disorders.

Six months later only bulimic episodes were reported, with no neurologic symptoms or further weight loss. Metabolic control although improved, was still poor (mean HbA1c: 8.7%). NCS was repeated after one year. Abnormal electrophysiologic parameters in the peroneal nerve slightly improved (SNAP: 5.1mV, SCV: 38.6msec), however abnormal and marginal values were also recorded in sural nerve (SNAP: 5.5uV, SCV: 40msec).

The teenager presented no other indices of microvascular complications.

	First NCS	*Second NCS*	*Normal values*
Peroneal nerve			
SL (msec)	3.98	3.9	<4.4
SNAP (μV)	2.7	5.1	≥6
SCV (m/sec)	35	38.5	≥40
DML (msec)	4.1	3.8	<6.5
CMAP (mV)	7.8	5.8	≥2
MCV (m/sec)	50	51	≥44
Sural nerve			
SL (msec)	3.04	2.94	<4.4
SNAP (μV)	10	5.5	≥6
SCV (m/sec)	50	40	≥40
Median nerve			
SL (msec)	2.42	2.36	<3.5
SNAP (μV)	31	28.9	≥20
SCV (m/sec)	53.7	55	≥50
DML (msec)	3.8	3.5	<4.4
CMAP (mV)	13.5	13.6	≥4
MCV (m/sec)	54	59	≥49

Table. Electrophysiologic parameters in the examined nerves of the female teenager, at the first (during the symptomatic period) and second examination (after 12 months).

Conclusion:

- Neurologic disorders have been described in patients with eating disorders, attributable to inadequate vitamin intake.
- Eating disorders in T1D teenagers have been associated to diabetic complications.
- APN may present in T1D patients during a period of acute weight loss due to poor vitamin intake and poor metabolic control, which are both involved in the pathogenesis of DN. Further development of DN, although subclinical, cannot be reversed unless optimal metabolic control is achieved and maintained in the long term. Therefore such patients must be closely followed in order to prevent further nerve damage.

ESPE 2015 757--P3
Diabetes 2
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DOI: 10.3252/pso.eu.54espe.2015
Poster presented at: ESPE 2015 BARCELONA European Society for Paediatric Endocrinology
PosterSessionOnline.com