Available online at www.sciencedirect.com

ScienceDirect

journal homepage: http://www.elsevier.com/locate/pjnns

Case report

Simultaneous acute shoulder arthritis and multiple mononeuropathy in a newly diagnosed type 2 diabetes patient – First case report

Dariusz Kotlęga [a,b,*], Monika Gołąb-Janowska [a], Grzegorz Zaborowski [c], Sylwester Ciećwież [d], Przemysław Nowacki [a]

[a] Department of Neurology, Pomeranian Medical University in Szczecin, Szczecin, Poland
[b] Aldemed Centrum Medyczne, Zielona Góra, Poland
[c] Magnetic Resonance Laboratory, Affidea Lubuskie Centrum Medyczne, Zielona Góra, Poland
[d] Department of Gynaecology and Urogynaecology, Pomeranian Medical University, Szczecin, Poland

ARTICLE INFO

Article history:
Received 25 December 2015
Received in revised form
8 July 2016
Accepted 13 July 2016
Available online 25 July 2016

Keywords:
Diabetes
Neuropathy
Shoulder arthritis
Diabetic neuropathy
Diabetic ketoacidosis

ABSTRACT

Diabetes is a common disorder that leads to the musculoskeletal symptoms such as the shoulder arthritis. The involvement of peripheral nervous system is one of the troublesome for the patients as it provokes chronic sensory symptoms, lower motor neuron involvement and autonomic symptoms. In the course of the disease there has been several types of neuropathies described.

A 41-year-old male patient was admitted to the internal medicine department because of the general weakness, malaise, polydypsia and polyuria since several days. The initial blood glucose level was 780 mg/dl. During the first day the continuous insulin infusion was administered. On the next day when he woke up, the severe pain in the right shoulder with limited movement, right upper extremity weakness and burning pain in the radial aspect of this extremity appeared. On examination right shoulder joint movement limitation was found with the muscle weakness and sensory symptoms in the upper limbs. The clinical picture indicated on the right shoulder arthritis and the peripheral nervous system symptoms such as the right musculocutaneous, supraspinatus, right radial nerve and left radial nerve damage.

We present a first case report of simultaneous, acute involvement of the shoulder joint and multiple neuropathy in a patient with newly diagnosed type 2 diabetes, presumably in the state of ketoacidosis.

* *Corresponding author at*: Department of Neurology, Pomeranian Medical University, Unii Lubelskiej 1, 71-252 Szczecin, Poland, Tel.: +48 914253251; fax: +48 914253260.
E-mail address: dkotlega@poczta.onet.pl (D. Kotlęga).

http://dx.doi.org/10.1016/j.pjnns.2016.07.003
0028-3843/

Introduction

In the diabetic patients there is higher risk of musculoskeletal symptoms, such as the shoulder arthritis called ''frozen shoulder'' that is an orthopedic disorder connected with the presence of diabetes. The frequency in diabetic patients ranges from 6.7% to 31.8%, while in non-diabetics is observed in 2–3% [1,2]. There is a strong association between risk of shoulder capsulitis (SC) and the duration of diabetes. In diabetic patients it is also more often bilateral. There has been shown correlation between SC and microvascular complications such as retinopathy and autonomic neuropathy [3].

The musculoskeletal complications in diabetes result from prolonged hyperglycemia that activates two metabolic cascades: enzymatic (throughout aldolase reductase) and non-enzymatic (throughout glication). Non-enzymatic glication of type IV colagen leads to increase of arterial resistance that decreases tissue perfusion and causes nerve hypoxia [2].

The diabetic neuropathy contains several types of clinical manifestations. The pathogenesis is linked to hyperglycemia and subsequent metabolic and ischemic changes, but also compressive and inflammatory etiology is proposed. The most common type of diabetic neuropathy is symmetrical, distal, sensorimotor polyneuropathy. Other types of diabetic neuropathy include lumbosacral radiculoplexus neuropathy (diabetic cachexia), neuropathies with involvement of trunk, cranial nerves, mononeuropathies and autonomic neuropathy. There are also distinct types of neuropathies such as ''neuropathy after ketoacidosis'' and diabetic radiculoplexus neuropathies (DRPN). The DRPN neuropathies consist of diabetic cervical radiculoplexus neuropathy (DCRPN), diabetic thoracic radiculoneuropathy (DTR) and diabetic lumbosacral radiculoplexus neuropathy (DLRPN) [4].

There has been only 2 patients presented with SC and neuropathy as a cervical radiculopathy [5]. To our knowledge there has not been presented cases with an acute, simultaneous shoulder inflammation and multiple mononeuropathy in the new onset diabetic patient with ketoacidosis.

Case presentation

A male, 41-year old Caucasian patient was admitted to the internal medicine department in the west part of Poland, because of high blood glucose level (780 mg/dl) detected by the general practitioner. The measurement was performed because of general weakness, malaise, polydypsia and polyuria that had been present for several days before. The initial laboratory results are listed in Table 1, the BMI was 28.5. During the first day after admission he was on the insulin pump with continuous insulin infusion. Till the next morning the blood glucose level of 200 mg/dl was reached and insulin infusion terminated. At this time, new symptoms appeared, such as the severe pain in the right shoulder with limited movement, right upper extremity weakness and burning pain within the radial aspect of this extremity. The exact time of the onset is not known, because patient woke up with these symptoms. After 7 days the weakness of left arm was noticed. There was number of additional tests performed. The abdominal ultrasonography showed moderate hepatic steatosis. The brain MRI detected bilaterally multifocal leukodystrophic lesions in the deep white matter. Other tests showed no abnormalities: cervical spine CT, the X-ray and ultrasonography of right shoulder, the right upper extremity Doppler ultrasonography. The concomitant medical history included hypertension and previous, distant glomerulonephritis. The orthopedics, rheumatologist and neurologist consultations were made, without establishing any diagnosis. After 1 week patient was sent home with drugs prescribed, including insulin and with advice to undergo further neurological diagnostics.

One month later patient visited the neurology outpatient clinic. The main complaints were pain with movement limitation in the right shoulder, weakness of the distal parts of the upper extremities with predominance on the right, burning pain radiating throughout radial aspect of the right upper extremity. On examination: muscle wasting in the right upper extremity within supraspinatus, subscapularis, teres major, brachial muscles, almost immobile right shoulder joint with 20° abduction, 30° flexion and 30° extension (Fig. 1). Muscle strength in the right elbow joint was decreased in the flexion movement. The strength in the right upper extremity girdle was difficult to assess, because of the severe passive and active movement limitation. There was also severe pain while shoulder movement. Flexion of the right wrist was 2 points in MRC (Medical Research Council) scale, extension 1 point. Decreased sensation of the radial aspect of right forearm and wrist was found. In the left upper extremity there was weakness of the wrist extension of 2 points MRC scale and decreased sensation over the skin area between the thumb

Table 1 – Initial blood and urine laboratory test results.

	Parameter	Result		Parameter	Result
1.	Creatinine	1.58 mg/dl	11.	CRP	12.98 mg/l
2.	Urea	44.5 mg/dl	12.	pH	7.36
3.	Natrium	132.8 mmol/l	13.	PCO2	29.4 mmHg
4.	Potassium	5.5 mmol/l	14.	HCO_3^-	16.2 mmol/l
5.	Glucose	678.22 mg/dl	15.	Urine density	1.015
6.	Alanine aminotranferase	215.84 U/l	16.	Urine pH	5.5
7.	Aspartate aminotransferase	104.77 U/l	17.	Urine glucose	2014 mg/dl
8.	TSH	1.28 uIU/ml	18.	C-peptide	3.56 ng/ml
9.	Triglycerides	311 mg/dl	19.	Insulin	3.3 uIU/ml
10.	LDL cholesterol	67.4 mg/dl	20.	HbA1c	15.8%

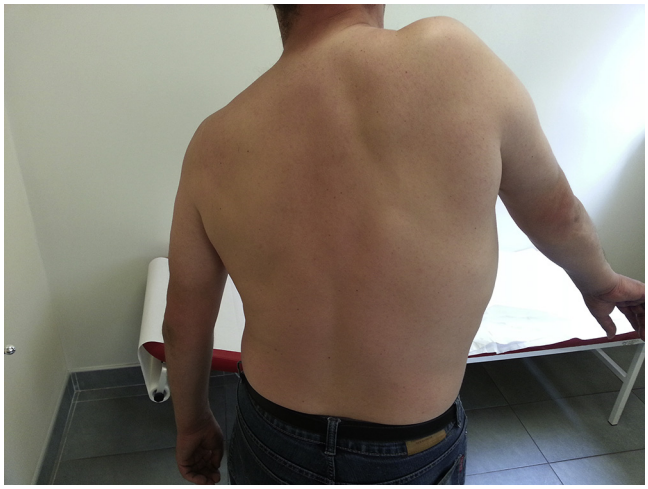

Fig. 1 – The image of a patient presenting the range of abduction movement.

and index finger. Additional tests were planned, ketoprofen 100 mg once daily and amitriptyline 50 mg once daily were prescribed, rehabilitation was advised.

During the outpatient elective diagnostics, the nerve conduction studies and MRI of right shoulder joint were performed 14 weeks after the onset of the symptoms. The shoulder MRI showed fluid in the tendon of biceps muscle and small amount in the joint capsule (Figs. 2 and 3), as well as thickened and heterogeneously increased density in the tendon of supraspinatus muscle (Fig. 4). The initial motor and nerve conduction study showed multifocal damage of the peripheral nervous system i.e. right musculocutaneous, right suprascapular nerves and left radial nerve (Tables 2 and 3). Right radial nerve was not tested at that time, but clinical picture indicated also on right radial nerve damage. The muscle records were not performed.

Patient was controlled at week 18 since the onset with gradual improvement of shoulder joint mobility, slight improvement in the muscle strength and sensory symptoms. He was undergoing rehabilitation and was taking prescribed

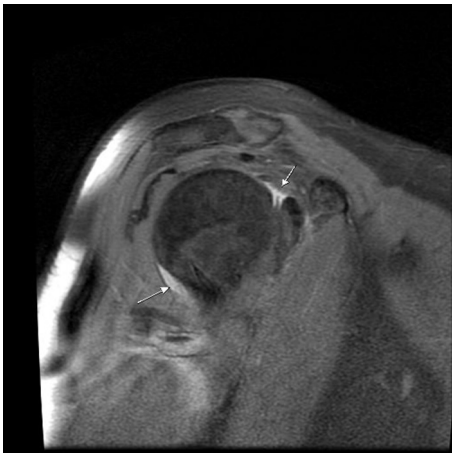

Fig. 2 – The MRI of right shoulder (PD FS).

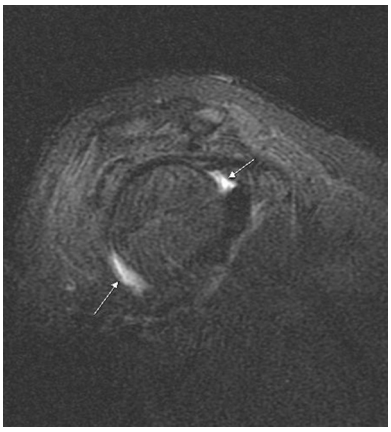

Fig. 3 – The MRI of right shoulder (STIR).

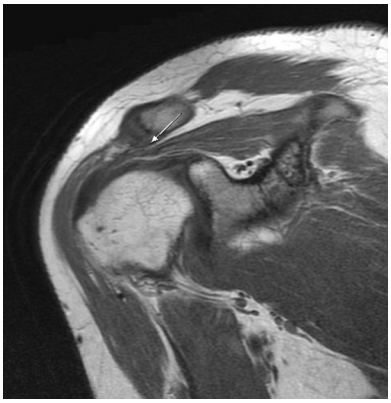

Fig. 4 – The MRI of right shoulder (T1).

drugs. Patient was referred to an orthopedics because of the right shoulder joint inflammation.

Next visit took place 6 months after the onset. The MRI of the right brachial plexus made 6 months after the onset was normal. Patient was not qualified for the surgical treatment. Gradual improvement of right shoulder mobility and muscle strength were achieved following the continuous rehabilitation. The shoulder joint was no longer painful. Neuralgic pain exacerbated on the radial aspect of the right forearm and wrist, sensation in the right arm came to normal. Diclofenac 75 mg twice daily and venlafaxin 75 mg once daily were prescribed instead of ketoprofen and amitriptyline. 16 months since the onset almost full motion range in the right shoulder joint was restored, the muscle strength significantly improved with the strength of 4 points MRC in the right wrist extension and 3 points on the left. The painful paresthesias resolved with only the hyposthesia persistent on the right side. The follow-up neurological examination was consistent with the electrophysiological studies that are presented in Tables 2–4.

Discussion

We presented a simultaneous onset of the right shoulder joint inflammation and multifocal mononeuropathy during the intensive insulin treatment of severe hyperglycemia in the

Table 2 – Sensory nerve conduction study.

Nerve/site	Record number (I-initial, II-follow-up)	Latency (ms)	Peak amplitude (uV)	Velocity (m/s)
Left median – digit II (palm)	I	2.55	8.6	47.8
	II	3.13	8.4	51.0
Left median – digit II (palm)	I	NT	NT	NT
	II	3.01	1.74	52.5
Left ulnar – digit V (wrist)	I	2.3	4.1	44.3
	II	3.0	3.4	46.1
Right ulnar – digit V (wrist)	I	2.1	6	47.6
	II	2.61	4.7	53.5
Left radial – thumb (forearm)	I	2.3	6.6	52.2
	II	2.0	3.2	52.4
Right radial – thumb (forearm)	I	NT	NT	NT
	II	–	–	–

NT, not tested; –, no response.

Table 3 – Motor nerve conduction study.

Nerve/site	Record number (I-initial, II-follow-up)	Latency (ms)	Peak amplitude (uV)	Velocity (m/s)
Left median (wrist)	I	2.8	13	
	II	3.63	10.6	
Left median (elbow)	I	8.15	12.7	52.3
	II	8.75	10.6	47.9
Left median (axilla)	I	10.95	12.7	57.1
	II	10.1	10.8	59.3
Right median (wrist)	I	2.8	7.6	
	II	3.37	9.7	
Right median (elbow)	I	7.85	7.8	52.5
	II	8.18	9.6	49.9
Right median (axilla)	I	11.35	6.5	50
	II	9.50	9.5	53
Left ulnar (wrist)	I	2.35	9.7	
	II	2.71	14.2	
Left ulnar (elbow)	I	7.65	7.2	55.7
	II	7.92	12.8	43.2
Left ulnar (axilla)	I	11.7	6	59.3
	II	9.47	12.9	51.6
Right ulnar (wrist)	I	2.15	6.3	
	II	2.73	11.5	
Right ulnar (elbow)	I	7.2	6.5	55.4
	II	6.87	10.7	53.1
Right ulnar (axilla)	I	12.1	7.8	49
	II	9.22	10.4	57.7
Left radial (forearm)	I	1.6	1.1	
	II	–	–	
Left radial (upper arm)	I	4.25	2	45.3
	II	–	–	–
Right radial (forearm)	I	NT	NT	
	II	1.76	11.0	
Right radial	I	NT	NT	NT
	II	4.65	11.8	48.1
Right radial	I	NT	NT	NT
	II	6.02	12.7	74.5
Right musculocutaneous – biceps (axilla)	I	1.8	1.1	
	II	NT	NT	
Right musculocutaneous – biceps (EP)	I	6.05	2	45.5
	II	4.23	6.0	
Right suprascapularis – infrascapularis, supraspinatus (EP – Infraspinatus)	I	2.6	1	46.4
	II	2.03	6.6	

NT, not tested; –, no response.

Table 4 – F wave records.

Nerve/site	F wave minimal latency (ms)	Persistence (%)
Left median (wrist – abductor pollicis brevis)	24.6	100
Right median (wrist – abductor pollicis brevis)	24.9	91.7
Right peroneal II (ankle – extensor digitorum brevis)	56.9	43.8
Right tibial (ankle – abductor hallucis)	59.7	100
Left ulnar (wrist – abductor minimi digiti)	25.2	100
Right ulnar (wrist – abductor minimi digiti)	25.4	100

new onset diabetes. There were no measurements of ketones made, but the clinical symptoms and other laboratory tests indicate on mild ketoacidosis state. The peripheral nervous system symptoms involved right musculocutaneous, supraspinatus, right radial nerve and left radial nerve damage. The limitation of our description is the fact, that initial muscle and extended nerve conduction studies were not performed. The follow-up electrophysiological studies indicated on the improvement in right suprascapular and musculocutaneous nerves, persistent damage to right radial nerve (initially detected only in physical examination, no electrophysiological testing was performed) and worsening within the left radial nerve. The minimal F wave latency records in the upper limbs were within the normal range, whereas in the lower limbs there was a prolonged latency and decreased persistence detected suggesting the spinal radiculopathy. The coincidence of chronic neuropathy with diabetes is common, but in a presented case the symptoms appeared with accompanying shoulder joint symptoms, suddenly, during the rapid hypoglycemic therapy of mild ketoacidosis in a newly diagnosed diabetes. To our knowledge, such a characteristics of symptoms in described for the first time.

The patients with a diabetic ketoacidosis and an acute neuropathy, but without shoulder joint involvement were reported. There is a case report when 1 week after the introduction of oral hypoglycemic treatment in a newly diagnosed diabetic patient sudden neuropathy symptoms appeared. The initial blood glucose level was 381 mg/dl. The peripheral nerve symptoms were axonal axillary and musculocutaneous neuropathy on the right side and panplexopathy on the left, with bilateral moderate to severe muscle weakness and mild hypoaesthesia. Of note may be the concomitant medical history with previous poliomyelitis and previously present polyneuropathy of lower extremities [6]. In a case report there was bilateral brachial plexopathy described during the diabetic ketoacidosis and intensive insulin treatment. A patient described with an acute brachial plexopathy associated with diabetic ketoacidosis had initial blood glucose level of 700 mg/dl with no previous history of diabetes. The symptoms of brachial plexus damage appeared 2 days after the onset of ketoacidosis [7].

Another case report described a patient with 13-year-history and well controlled type 2 diabetes on insulin therapy for 5 preceding years. The onset of the symptoms was not acute, but gradual with progressive left arm weakness that evolved for more than 3 months after the onset of pain and stabilized thereafter. Right arm weakness developed over the preceding 3 months and has continued to progress. Electromyography and nerve conduction studies revealed bilateral upper-trunk brachial plexopathy [8]. There are important differences between this and our patient within the gradual onset of the symptoms, previous chronic treatment of diabetes and distribution of neuropathy.

There is a case report of a newly onset type 1 diabetes with blood glucose level of 629 mg/dl, subsequent ketoacidosis and mononeuropathy of peroneal nerve [9]. Another patient with de novo type 1 diabetes in diabetic ketoacidosis developed simultaneous intracerebral hemorrhage with the left ulnar nerve palsy, ulnar, median, and radial nerve palsies on the right on day 7 since the diabetic ketoacidosis and comatose state for first the 24 h [10]. Another patient – 10-year-old girl was diagnosed of de novo type 1 diabetes with the initial blood glucose level of 629 mg/dl and the electrophysiological study indicating on a lesion of the peroneal nerve [11]. In a 9-year-old boy with newly diagnosed type 1 diabetes and ketoacidosis the acute peripheral nervous system damage was noticed i.e. complete lesion of the right radial, the left common peroneal nerves and partial lesion of the right common peroneal and sural nerves [12].

There has been only 2 cases reported with simultaneous shoulder arthritis and neuropathy in a diabetic patient. In a first case the left arm weakness with left shoulder movement limitation were demonstrated 3 months after type 2 diabetes diagnosis and oral hypoglycemic agent were introduced. The radiographic and clinical diagnosis of shoulder capsulitis was made with C5 radiculopathy on the left side, which was not resulting from spine pathology. The second patient had previous history of type 2 diabetes with oral hypoglycemic drugs and C5 radiculopathy on the right with ipsilateral shoulder capsulitis. There is no available information if the symptoms had chronic or acute onset [5].

There is a question raised, what may be the mechanism of such an acute, diffused damage to the peripheral nervous system. The diabetic neuropathy is proved as a consequence of metabolic changes including polyol pathway flux, increased advanced glycosylation, elevated oxidative stress and impaired fatty acid metabolism [13]. Diabetic ketoacidosis is a proinflammatory condition with vascular endothelial perturbation and dysregulation of the coagulation system features associated with abnormal levels and activities of several coagulation factors, including platelets, which result in a procoagulant state. Additional factors contributing to the procoagulant state are abnormalities in blood volume, blood viscosity, cerebral autoregulation and blood flow, while vascular injury may be a result of oxidative injury and ischemia related to systemic hypoperfusion, vascular dysregulation, or cerebral edema [14]. The neurological impairment may be connected with the functional neural lesions due to

vasa nervorum thrombosis and prolonged ischemia [12]. The immune system response in generating the neuropathy may be suggested on the basis of two patients with a Guillain-Barre syndrome developed just after treating the ketoacidosic comatose state in new onset diabetes [15]. The pathogenesis of DLRPN is the ischemic injury caused by vasculitis [16]. Another explanation may be the neuronal damage due to hyperglycemia metabolic effects, such as metabolic acidosis, hyperosmolality, dehydration or electrolyte imbalance [17]. It is also possible that both ischemic and inflammatory component is involved in this process. The nerve biopsy in DCRPN shows ischemic injury and increased inflammation [18]. The immune system activation play the key role in the LADA diabetes (Latent Autoimmune Diabetes of Adults), but in our patient the diagnosis of type 2 diabetes was established [19].

An explanation of the simultaneous shoulder joint inflammation with neuropathy is not clear. The most convincing pathogenetic background may be the metabolic effects or inflammation. In a biopsy-based study of the elderly diabetic neuropathy patients, authors suggested that the pathogenesis is related to the pre-capillary blood vessel involvement with a secondary inflammatory response [20]. Inflammatory agents may play role in the neuropathy and shoulder arthritis. Other explanation is also possible, especially that in the SC patients the neuronal proteins expression is increased and new nerve fibers are found. There was demonstrated increased expression of the nerve growth factor receptor p75 and CD34 – an endothelial cell marker, thenprotein gene product 9.5 (PGP9.5) – general nerve marker and the growth associated protein 43 (GAP43) that is a nerve growth marker. These results indicate on neoinnervation and neoangiogenesis in SC, which may be essential for explanation the pathogenesis of simultaneous joint and nerve damage [21].

Another issue is to establish the type of neuropathy in terms of lesion distribution. Symptoms observed in our patient suits the most to the ''neuropathy after ketoacidosis'' which is very rare condition with clinical picture of multiple mononeuropathies [4]. Less likely there might be the diagnosis of DCRPN which leads to moderate motoric deficits with improvement at follow-up. Upper, middle and lower brachial plexus segments are involved equally and over half of patients had at least one additional body region affected (contralateral cervical, lumbosacral, thoracic or less common isolated peripheral nerve alone) [18].

Presented case was the first patient with simultaneous multiple mononeuropathy, probably the ''neuropathy after ketoacidosis'' type and shoulder arthritis. Biopsy studies would be useful to unearth the pathogenesis, which was not performed as well as extended initial electrophysiological studies, but there were the follow-up electrophysiological records performed 16 months since the onset that confirmed clinical diagnosis. Our description may be important in further studies in the field of diabetic neuropathy and joint involvement in diabetics. Such an unusual presentation has also the educational value for neurologists.

Conflict of Interest

None declared.

Acknowledgment and Financial Support

None declared.

Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

REFERENCES

[1] Kiani J, Goharifar H, Moghimbeigi A, Azizkhani H. Prevalence and risk factors of five most common upper extremity disorders in diabetics. JRHS 2014;14:93–6.
[2] Parada-Turska J, Majdan M. The musculoskeletal system in diabetic patients. Postepy Hig Med Dosw (online) 2005;59:236–44.
[3] Arkkila PET, Kantola IM, Viikari JSA, Ronnemaa T. Shoulder capsulitis in type I and II diabetic patients: association with diabetic complications and related diseases. Ann Rheum Dis 1996;55:907–14.
[4] Tracy JA, Dyck PJB. The spectrum of diabetic neuropathies. Phys Med Rehabil Clin N Am 2008;19:1–26. http://dx.doi.org/10.1016/j.pmr.2007.10.010
[5] Seo JH, Park SH. Diabetic cervical radiculopathy with adhesive capsulitis of the shoulder. Yonsei Med J 2003;44:1114–8.
[6] Pica EC, Verma KK. Bilateral brachial plexopathy as an initial presentation in a newly-diagnosed, uncontrolled case of diabetes mellitus. Singapore Med J 2008;49: e29–32.
[7] Santillan CE, Katirji B. Brachial plexopathy in diabetic ketoacidosis. Muscle Nerve 2000;23:271–3.
[8] Muley SA, Parry GJ, Ercan-Fang NG. Isolated bibrachial plexopathy in a patient with type 2 diabetes. Diabetes Care 2005;28:2591–2.
[9] Rangel MA, Baptista C, Santos F, Real MV, Campos RA, Leite AL. Acute mononeuropathy in a child with newly diagnosed type 1 diabetes mellitus. J Pediatr Endocrinol Metab 2015;28 (3–4):341–4. http://dx.doi.org/10.1515/jpem-2014-0049
[10] Atkin SL, Coady AM, Horton D, Sutaria N, Sellars L, Walton C. Multiple cerebral haematomata and peripheral nerve palsies associated with a case of juvenile diabetic ketoacidosis. Diabet Med 1995;12:267–70.
[11] Rangel MA, Baptista C, Santos F, Real MV, Campos RA, Leite AL. Acute mononeuropathy in a child with newly diagnosed type 1 diabetes mellitus. J Pediatr Endocrinol Metab 2015;28(3–4):341–4. http://dx.doi.org/10.1515/jpem-2014-0049
[12] Bonfanti R, Bognetti E, Meschi F, Medaglini S, D'Angelo A, Ghiumello G. Disseminated intravascular coagulation and severe peripheral neuropathy complicating ketoacidosis in a newly diagnosed diabetic child. Acta Diabetol 1994;31:173–4.
[13] Cameron NE, Cotter MA. Metabolic and vascular factors in the pathogenesis of diabetic neuropathy. Diabetes 1997;46 (Suppl 2):S31–7. http://dx.doi.org/10.2337/diab.46.2.S31
[14] Foster JR, Morrison G, Fraser DD. Diabetic ketoacidosis-associated stroke in children and youth. Stroke Res Treat

2011;2011(February):219706. http://dx.doi.org/10.4061/2011/219706

[15] Rouanet-Larriviere M, Vital C, Arne P, Favarel-Garrigues JC, Gin H, Vital A. Guillain-Barré syndrome occurring in two women after ketoacidosic comatose state disclosing an insulin-dependent diabetes mellitus. J Peripher Nerv Syst 2000;5:27–31.

[16] Dyck PJ, Norell JE, Dyck PJ. Microvasculitis and ischemia in diabetic lumbosacral radiculoplexus neuropathy. Neurology 1999;53:2113–21.

[17] Tsai SL, Hadjiyannakis S, Nakhla M. Hyperglycemic hyperosmolar syndrome at the onset of type 2 diabetes mellitus in an adolescent male. Paediatr Child Health 2012;17:24–6.

[18] Massie R, Mauermann ML, Staff NP, Amrami KK, Mandrekar JN, Dyck PJ, et al. Diabetic cervical radiculoplexus neuropathy: a distinct syndrome expanding the spectrum of diabetic radiculoplexus neuropathies. Brain 2012;135:3074–88.

[19] Litwinowicz M, Rogowicz A. Latent autoimmune diabetes of adults – what do we know today? Diabet Prakt 2010;11 (3):84–8.

[20] Said G, Lacroix C, Lozeron P, Ropert A, Planté V, Adams D. Inflammatory vasculopathy in multifocal diabetic neuropathy. Brain 2003;126:376–85.

[21] Xu Y, Bonar F, Murrell GA. Enhanced expression of neuronal proteins in idiopathic frozen shoulder. J Shoulder Elbow Surg 2012;21:1391–7.