The Egyptian Rheumatologist (2016) 38, 233–239

Egyptian Society of Rheumatic Diseases

The Egyptian Rheumatologist

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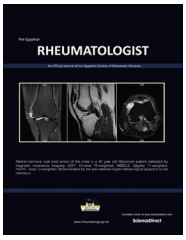

ORIGINAL ARTICLE

Clinical and electrophysiological study of peripheral and central neuromuscular changes in connective tissue diseases in children

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Received 24 August 2015; accepted 2 September 2015
Available online 23 October 2015

KEYWORDS

Juvenile connective tissue disease;
Neurological abnormalities;
Electrophysiological changes

Abstract *Introduction:* Children with juvenile connective tissue diseases (JCTDs) may have a wide variety of clinical features ranging from fever or a simple arthritis to complex multisystem autoimmune diseases.

Aim of the work: To study clinical and electrophysiological peripheral and central neuromuscular changes in children with connective tissue diseases.

Patients and methods: Thirty children with different JCTDs were enrolled. Clinical and neurological examination and laboratory investigations were done. Electrophysiological evaluation was performed and included: peripheral nerve conduction studies, late responses, somatosensory evoked potential and electromyography.

Results: Twenty patients had juvenile idiopathic arthritis (JIA) (66.7%), 8 patients had juvenile systemic lupus erythematosus (JSLE) (26.7%), one patient had juvenile systemic sclerosis (JSScl), and one patient had juvenile overlap syndrome (JSScl and polymyositis). Clinical neurologic abnormalities were present in 3 patients (ulnar neuropathy, median neuropathy and polymyositis). Electrophysiological abnormalities were detected in 18 patients (clinical in 3 and subclinical in 15 patients) and included ulnar entrapment neuropathy, median axonal neuropathy, demyelinating sensory motor polyneuropathy, deep peroneal nerve entrapment at the ankle (anterior tarsal tunnel syndrome), prolonged posterior tibial somatosensory evoked potential latency and prolonged H reflex latency not explained by peripheral neuropathy, increased H/M ratio and myopathic motor units. The most common electrophysiological abnormalities were present in patients with JSLE.

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Peer review under responsibility of Egyptian Society of Rheumatic Diseases.

http://dx.doi.org/10.1016/j.ejr.2015.09.002

Conclusion: Clinical neurological abnormalities are not common in JCTDs whereas subclinical neurological abnormalities are common findings. Juvenile systemic lupus erythematosus had the most common abnormalities among JCTDs. Polyneuropathy in JIA is commonly of demyelinating type. Entrapment neuropathy is less frequent than in adults.

1. Introduction

Children with juvenile connective tissue diseases (JCTDs) may have a wide variety of clinical features ranging from fever or a simple arthritis to complex multisystem autoimmune diseases [1]. Juvenile connective tissue diseases (JCTDs) are autoimmune multisystem inflammatory disorders including juvenile idiopathic arthritis (JIA), juvenile systemic lupus erythematosus (JSLE), juvenile systemic sclerosis (JSScl), juvenile dermatomyositis (JDM) and polymyositis, juvenile mixed connective tissue disease (JMCTD), juvenile Sjögren's syndrome (JSS) and vasculitis [2]. Different organs other than the musculoskeletal system may be involved including skin, kidneys, cardiopulmonary system, gastrointestinal and nervous systems [3].

Neurologic manifestations of rheumatic disorders can arise in both primary and secondary fashion [4]. That is, the antibodies or cellular immune elements responsible for the underlying disease can directly attack and injure nerves, muscle, brain, spinal cord, and sensory organs. On the other hand, innocent bystander effects of such rheumatic disease accompaniments as the hypercoagulable state, inflammation of the blood vessel wall, immune complex deposition and side effects of medications used in the treatment of rheumatic disease also take their toll on the nervous system [5]. Electrophysiological studies including nerve conduction studies (NCSs), electromyography (EMG) and somatosensory evoked potentials (SEPs) are most often used to diagnose disorders of the peripheral nervous systems (PNS) and central nervous systems (CNS) and provide valuable information about the underlying pathology [6,7].

The aim of this study was to study the clinical and electrophysiological peripheral and central neuromuscular changes in children with connective tissue diseases (CTDs) and to describe the neurologic complications of childhood rheumatic disease.

2. Patients and methods

Thirty children with different JCTDs (JIA, JSLE, JSScl, JDM and polymyositis, juvenile overlap syndrome and JSS) attending the pediatric rheumatology outpatient in Al-Shatby children university hospital were enrolled. Exclusion criteria included the presence of other neurological diseases, endocrinal, chronic infection, malignancy and heritable connective tissue diseases. Clinical and neurological examination and laboratory investigations were done. Thirty healthy children of matched age and sex were included as a control group. The study was approved by the local ethics committee of the Faculty of Medicine, Alexandria University and the patients gave an informed consent before inclusion in the study.

Electrophysiological evaluation was performed and included:

1. Peripheral NCSs: Nerve conduction studies were carried out using NIHON KOHDEN (Neuropack 2) electrophysiologic apparatus. All recordings of action potentials were carried out by surface electrodes (8 mm) in diameter and a ground electrode was placed between the stimulating and recording electrodes. Stimulation was carried out using bipolar stimulator having a production current ability of 50 mA. The filter setting was between 2 Hz and 10 kHz. The following nerves were studied unilaterally; sural nerve (sensory study), posterior tibial and deep peroneal nerves (motor study), radial nerve (sensory study), median and ulnar nerves (motor and sensory studies). Involvement of one peripheral nerve only was defined as mononeuropathy. Abnormality in 2 or more nerves was defined as a peripheral neuropathy electrophysiologically [8].
2. Late responses: *F* wave of median and ulnar nerves and the soleal H-reflex were performed unilaterally.
3. SEPs: SEPs of median and posterior tibial nerves with cortical recording were done.
4. EMG: Standard concentric needle EMG of gluteus medius and tibialis anterior was performed unilaterally.

Statistical analysis: Student *t* and Mann–Whitney tests were used to determine statistical differences between patients and controls as regards the values of NCSs and SEPs. Normal values for our laboratory were obtained from control children and abnormal values were defined as 2 standard deviations above/below the normal mean. Significant values were considered at $p < 0.05$.

3. Results

Twenty-two patients were girls (73.3%), and 8 patients (26.7%) were boys. Their age ranged from 6 to 16 years with a mean of 12.37 ± 2.92 years. There was no statistically significant difference between patients and controls as regards age ($p = 0.35$) and sex ($p = 0.27$). Twenty children had JIA, 8 had JSLE, one had JSScl and another had juvenile overlap syndrome.

Patients had involvement of various systems; 29 patients (96%) had articular manifestations, and 22 (73%) developed extra-articular extra-neurological manifestations during the course of disease (skin manifestations, subcutaneous nodules, fever, headaches, hypertension, diabetes mellitus, abdominal pain and fatigue). Only 3 patients (10%) developed neurological abnormalities; two of them had JSLE associated with lupus nephritis (one with ulnar mononeuropathy who had decreased sensation on ulnar nerve distribution of the right

hand and forearm associated with wasting of small hand muscles, partial claw hand deformity and grade 2 weakness of the hypothenar muscles, interossei and adductor pollicis muscles; the other one with median mononeuropathy who had decreased sensation on median nerve distribution of the right hand associated with grade 3 weakness of the thenar muscles. Phalen's and Tinel's tests were negative) and the 3rd had overlap syndrome (JSScl and JDM), showed bilateral symmetrical proximal muscle weakness of the 4 limbs associated with hardening of skin and subcutaneous calcinosis. All patients received non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids, 19 received methotrexate (MTX), 6 received hydroxychloroquine (HCQ), 5 received azathioprine (AZA), one received sulfasalazine (SAS), one received mycophenolate mofetil (MMF) and another received cyclophosphamide (CYC). Treatment was received at the optimal doses according to weight and disease activity.

As regards laboratory findings, 15 patients (50%) showed abnormal blood picture (anemia, leukocytosis, leucopenia, thrombocytosis, thrombocytopenia), 14 patients (46%) had increased acute phase reactants and 7 patients had proteinuria. Creatine phosphokinase (CPK) level was normal in all studied patients.

The results of motor conduction studies in patients and controls are demonstrated in Table 1. Statistically significant differences were detected between patients and controls as regards the distal latency and conduction velocity of posterior tibial and deep peroneal nerves as well as the ulnar nerve distal latency and amplitude. The results of sensory conduction studies in patients and controls are demonstrated in Table 2. Statistically significant differences were found as regards the distal latency, conduction velocity and amplitude of sural nerve as well as the distal latency of the median and ulnar nerves.

A significant difference was found between patients and controls as regards the H reflex latency (25.57 ± 3.39 vs 22.01 ± 3.25, $p < 0.001$). Significant differences were recognized as regards posterior tibial SEP latency and amplitude (Table 3).

Electromyographic abnormalities were detected in 10 patients (33%). One of them had abnormal resting potentials in the form of complex repetitive discharge, small short polyphasic motor units and early recruitment (consistent with electrophysiologic findings of polymyositis). The other 9 patients had small short polyphasic motor units consistent with myopathy with no abnormal resting activities, with normal recruitment.

By comparing the values of electrophysiological studies obtained from patients to the cut off values obtained from controls, electrophysiological abnormalities were detected in 18 patients (clinical in 3, subclinical in 15).

Table 1 Motor nerve conduction studies of the median, ulnar, posterior tibial and deep peroneal nerves in juvenile connective tissue diseases children and control.

Median (range) Mean ± SD	Motor NCSs			
	Latency (ms)	Amplitude (mV)	NCV (m/s)	*F* latency (ms)
Median nerve				
Patients ($n = 30$)	3 (1.9–4.7)	11.5 (1.2–23)	57 (39–79)	22.4 (16–27)
	3.04 ± 0.6	11.6 ± 4.7	56.6 ± 9.1	22.4 ± 2.7
Control ($n = 30$)	3.4 (2.4–3.7)	10.5 (6.3–19)	52.5 (48–68)	23 (19.5–26)
	3.2 ± 0.4	11.1 ± 3.9	53.7 ± 5.02	22.8 ± 1.7
t	1.03	0.49	1.55	0.66
p	0.31	0.63	0.13	0.51
Ulnar nerve				
Patients ($n = 30$)	2.7 (1.6–5)	8.9 (0.8–17)	58.5 (42.5–92)	23 (16–27)
	2.7 ± 0.6	8.8 ± 3.4	59.98 ± 13.4	23 ± 2.7
Control ($n = 30$)	2.4 (1.9–2.8)	11.5 (6.8–18)	60.8 (50–79)	22.8 (19–26.6)
	2.4 ± 0.3	11.8 ± 2.7	61.9 ± 8.9	22.5 ± 2.1
t	2.45	3.82	0.65	0.79
p	**0.017**	**< 0.001**	0.52	0.43
Posterior tibial nerve				
Patients ($n = 30$)	3.8 (2.4–6.5)	19 (5.5–32)	46 (31–64)	
	3.9 ± 0.9	16.8 ± 7.3	46.98 ± 6.9	
Control ($n = 30$)	3.3 (2.1–4.6)	15.3 (5–23)	49.5 (45–62)	
	3.4 ± 0.6	15.1 ± 4.9	51.2 ± 5.1	
t or *Z*	2.66	1.01	2.69	
p	**0.01**	0.31	**0.009**	
Deep peroneal nerve				
Patients ($n = 30$)	4.1 (2.8–7)	2.7 (0.8–12)	49 (39–67)	
	4.3 ± 0.97	3.5 ± 2.4	49.03 ± 6.3	
Control ($n = 30$)	3.8 (2.5–5.1)	3 (2–7)	51.3 (45–60)	
	3.8 ± 0.7	3.4 ± 1.3	52.2 ± 4.9	
t	2.25	0.62	2.15	
p	**0.028**	0.54	**0.036**	

NCSs: nerve conduction studies. Bold values are significant at $p < 0.05$.

Table 2 Sensory nerve conduction studies of the median, ulnar, superficial radial and sural nerves in juvenile connective tissue diseases children and control.

Median (range) Mean ± SD	Sensory NCSs		
	Latency (ms)	Amplitude (μV)	NCV
Median nerve			
Patients (n = 30)	2.95 (2–4)	30 (14–108)	52 (34.3–96)
	2.97 ± 0.5	39.95 ± 26.7	54.5 ± 12.3
Control (n = 30)	2.4 (1.4–3.3)	46.5 (20.7–65)	50.5 (49–59)
	2.44 ± 0.45	43.6 ± 15.6	52.3 ± 3.4
t or Z	4.23	1.81	0.97
p	**< 0.001**	0.07	0.34
Ulnar nerve			
Patients (n = 30)	2.6 (1.8–3.8)	30 (0.5–108)	54 (31.3–80)
	2.47 ± 0.6	37.6 ± 24.04	55.2 ± 10.5
Control (n = 30)	2.05 (1.6–2.5)	36.5 (22–45)	51.5 (49–64)
	2.1 ± 0.3	35.97 ± 8.3	53.1 ± 4.5
t or Z	5.92	1.17	0.997
p	**< 0.001**	0.24	0.33
Superficial radial nerve			
Patients (n = 30)	2.2 (1.7–3.4)	30 (10–71)	53.5 (37–87)
	2.3 ± 0.4	32.6 ± 17.1	55.8 ± 11.7
Control (n = 30)	2.2 (1.7–3)	30 (16.8–45)	53.5 (49–55.6)
	2.3 ± 0.4	31.22 ± 8.6	52.7 ± 2.2
t	0.85	0.52	1.43
p	0.4	0.6	0.16
Sural nerve			
Patients (n = 30)	3.8 (2.2–5)	12.3 (3–90)	45 (33.3–60)
	3.6 ± 0.7	16.1 ± 15.7	45.02 ± 6.3
Control (n = 30)	2.4 (1.7–3.3)	20.2 (10–32)	49 (44–55)
	2.5 ± 0.5	21.2 ± 6.4	49.4 ± 4.1
t or Z	7.31	3.6	3.2
p	**< 0.001**	**< 0.001**	**0.002**

NCSs: nerve conduction studies. Bold values are significant at $p < 0.05$.

Table 3 Somatosensory evoked potential of median and posterior tibial nerves in juvenile connective tissue diseases children and control.

Median (range) Mean ± SD	Patients (n = 30)	Control (n = 30)	t or Z	p
Median nerve				
SEP latency (ms)	17 (15–20)	17 (15.5–19)	t = 0.43	0.67
	16.98 ± 1.1	16.9 ± 0.8		
SEP amplitude (μV)	7.65 (3–20)	3.56 (6.5–18)	t = 0.1	0.92
	8.8 ± 4.2	9.4 ± 3.5		
Posterior tibial n				
SEP latency (ms)	36.9 (30–43)	33.8 (30–37)	t = 2.07	**0.04**
	37 ± 3.5	34.8 ± 2.2		
SEP amplitude (μV)	4 (1–14)	6 (3.8–13)	Z = 3.33	**0.001**
	5 ± 3.2	6.4 ± 2.2		

SEP: somatosensory evoked potential. Bold values are significant at $p < 0.05$.

Patients with JSLE had the most common electrophysiologic abnormalities; seven out of 8 patients (87%) (2 clinical and 5 subclinical). Subclinical electrophysiological abnormalities in JSLE patients included demyelinating sensory polyneuropathy in one patient, prolonged posterior tibial SEP latency and prolonged H reflex latency (not explained by peripheral neuropathy) in 2 patients, increased H/M ratio in one patient and myopathic motor unit without any other electrophysiological abnormality in 2 patients.

Electrophysiologic abnormalities were detected in 10 out of 20 patients with JIA (50%). All had only subclinical findings in the form of demyelinating sensory motor polyneuropathy in 2 patients, right deep peroneal nerve (DPN) entrapment at the ankle (anterior tarsal tunnel syndrome) in one patient,

Table 4 Relationship between electrophysiological abnormalities and the drug received by the studied children with juvenile connective tissue diseases.

Medication *n* (%)	Electrophysiological abnormalities		χ^2	*p*
	Normal (*n* = 12)	Abnormal (*n* = 18)		
Prednisolone				
No	0 (0)	0 (0)	–	–
Yes	12 (100)	18 (100)		
NSAIDs				
No	2 (16.7)	8 (44.4)	2.5	0.24
Yes	10 (83.3)	10 (55.6)		
MTX				
No	3 (25)	8 (44.4)	1.17	0.44
Yes	9 (75)	10 (55.6)		
AZA				
No	11 (91.7)	14 (77.8)	1	0.62
Yes	1 (8.3)	4 (22.2)		
HCQ				
No	11 (91.7)	13 (72.2)	1.7	0.36
Yes	1 (8.3)	5 (27.8)		
SAS				
No	12 (100)	17 (94.4)	0.69	1
Yes	0 (0)	1 (5.6)		
MMF				
No	12 (100)	17 (94.4)	0.69	1
Yes	0 (0)	1 (5.6)		
CYC				
No	12 (100)	17 (94.4)	0.69	1
Yes	0 (0)	1 (5.6)		

NSAIDs: non-steroidal anti-inflammatory drugs; MTX: methotrexate; AZA: azathioprine; HCQ: hydroxychloroquine; SAS: sulfasalazine; MMF: mycophenolate mofetil; CYC: cyclophosphamide.

prolonged posterior tibial SEP latency without peripheral neuropathy in one patient and myopathic motor units without any other electrophysiological abnormality in 6 patients.

One patient with overlap syndrome (JSScl and polymyositis), showed electrophysiological evidences of polymyositis (complex repetitive discharge, myopathic motor units and early recruitment). The patient with JSScl did not show any electrophysiologic abnormalities.

No relationship was found between electrophysiological abnormalities and different parameters (disease duration, gap between disease onset and start of treatment and intake of prednisolone, NSAIDs, MTX, HCQ, AZA, CYC, MMF and SAS (Tables 4 and 5). In this work only one patient received CYC, another one received MMF and a third one received SAS. These 3 patients showed electrophysiological abnormalities.

4. Discussion

Peripheral and central neurological complications in adult patients with CTDs have been previously studied [9–11], but for children are still deficient.

In the current study clinical and electrophysiological studies were implemented to detect neurologic abnormalities in children with JCTDs. Clinical neurological abnormalities were present in 3 patients (10%), and subclinical neurologic abnormalities were present in 15 (50%). Both clinical and subclinical neurologic abnormalities were present mainly in JSLE patients. There were no clinical neuropathic abnormalities in the JIA patients or in the patients with JSScl or overlap. This finding is consistent with other studies which found that the nervous system is commonly affected in children with JSLE, and it was reported to occur in 22–95% [12] and is less prevalent in other JCTDs [13].

Patients with JSLE showed clinical neuropathic abnormalities in 25% of patients and subclinical electrophysiologic abnormalities in 62%. This finding is consistent with another study on adult systemic lupus erythematosus (SLE) patients that found clinical signs of peripheral neuropathy in 28% and abnormal electrophysiological findings in 56%. This clearly suggested that a sizable proportion of SLE patients have subclinical peripheral nerve disease [8].

Peripheral NCS abnormalities in JSLE patients were in the form of right ulnar entrapment mononeuropathy, right median axonal mononeuropathy and demyelinating sensory polyneuropathy. These results are of support to the findings of another study on JSLE patients reporting that peripheral nerve involvement is the most common presentation [14]. Peripheral neuropathy in JSLE patients develops due to vasculitic insult to vasa nervosum. There is Wallerian degeneration of nerve fibers secondary to ischemic infarction due to occlusion of blood vessels caused by leukocytoclastic vasculitis. In their study, sural nerve biopsy in JSLE neuropathy patients found that endoneurial immune complex deposition also plays an important role in the demyelinating process and axonal damage seen in peripheral neuropathy [14].

In 2 patients with JSLE, increased H-reflex latency was not associated with peripheral neuropathy; this electrophysiological finding is coincident with the study done for adult SLE patients that found prolonged or absent H reflex as common

Table 5 Comparison between children with juvenile connective tissue diseases and abnormal electrophysiological findings and those without as regards the disease duration and the gap between disease onset and start of treatment.

Median (range) Mean ± SD	Electrophysiological abnormalities		*Z*	*p*
	Normal (*n* = 12)	Abnormal (*n* = 18)		
Disease duration (month)	60 (3–96)	54 (12–102)	0.36	0.72
	57.8 ± 33.4	53.3 ± 30.4		
Gap between disease onset and start of treatment (month)	12 (0–24)	8 (0–90)	0.15	0.88
	9.9 ± 8.6	17.67 ± 24.1		

abnormal parameters. It was suggested that the proximal nerve segment may be prominently involved and underlying pathophysiological mechanism affecting the nerves does so in a patchy manner rather than through a dying back phenomenon [8].

As regards JIA patients, electrophysiologic abnormalities were detected in 50%. All had only subclinical findings, in the form of demyelinating polyneuropathy in 10%, right DPN entrapment at the ankle (anterior tarsal tunnel syndrome) in 5%, prolonged SEP latency without any other electrophysiologic abnormalities in 5% and myopathic motor units without any other electrophysiologic abnormalities in 30%. These findings differ from rheumatoid arthritis (RA) where polyneuropathy in adults is mainly of axonal type and entrapment neuropathies is the most common type of peripheral nerve disorders, that commonly affect the median nerve (carpal tunnel syndrome) [9,15,16].

In our study, no patient showed carpal tunnel syndrome. This finding is supported by a previous study done for children suffering from JIA, where it was found that, although wrist involvement in JIA is similar to that seen in adults, median nerves were electrophysiologically normal in JIA patients, either with or without wrist involvement [15]. It was speculated that increasing the carpal tunnel content due to the inflammatory process may be tolerated by more elastic and growing fibro-cartilaginous structures of the carpal tunnel in children with arthritis. Another factor would be the difference between the wrist deformities; being generally ulnar in JIA and being radial in RA [15].

In our study, one patient with JIA developed subclinical DPN entrapment under the inferior extensor retinaculum. This patient had JIA of polyarticular type with arthritis of both ankles and foot joints. A common site for DPN entrapment, along the dorsum of the foot, occurs in the region of the first and second tarsometatarsal joints as the medial branch of the DPN traverses a narrow fibro-osseous tunnel between the extensor hallucis brevis tendon and the deep fascia [17]. Entrapment occurs as the tendon crosses over the nerve. One of the known causes of DPN entrapment is arthritis of the talonavicular joint or at the first and second tarsometatarsal joints or tendinitis or hypertrophic muscle belly of extensor hallucis longus, extensor digitorum longus or tibialis anterior [18,19].

Prolonged SEP latency (not associated with peripheral neuropathy) was found in 4 patients, 2 of them had JIA and the other 2 had JSLE. This can be explained by a focal or generalized vasculopathy affecting the CNS [4].

The child with JSScl did not show any electrophysiological abnormalities. This finding comes in agreement with previous studies done for adults and children with SScl and they concluded that SScl is thought to be the least likely of the collagen vascular disorders to cause nervous system damage (peripheral or central) [20–22], only children with the coup-de-sabre form of localized scleroderma are at risk for CNS involvement [23].

The patient with overlap syndrome (JSScl and polymyositis) had objective neurologic abnormality. This is consistent with a previous study which found that when polymyositis occurs in children, it is often in the setting of overlap syndrome with other CTDs [24]. This child had no electrophysiologic abnormality on NCS but abnormalities were present on EMG study in the form of abnormal resting activities, myopathic motor units and early recruitment. This was revealed by other studies for children and adults with polymyositis where peripheral nerve and CNS involvement was rare in polymyositis and in dermatomyositis [25–27].

Myopathic motor unit action potentials were found in 9 patients (30%) (2 patients with JSLE and 7 patients with JIA). All of them were receiving corticosteroids, so these myopathic motor unit action potentials are mostly associated with corticosteroids use [28,29]. Myositis is distinguished from corticosteroid-related myopathy by demonstrating electromyographic spontaneous activity which reflects the ongoing disease activity [29,30].

A significant difference between patients and the control as regards the distal motor latency of the ulnar, posterior tibial and deep peroneal nerves and compound muscle action potential amplitude of ulnar nerve as well as motor conduction velocity of posterior tibial and deep peroneal nerves was observed. On the other hand, the sural nerve sensory conduction velocity and sensory nerve action potential amplitude showed a significant difference when compared with the control. These results were in accordance with previous studies done for adult patients with CTDs, which realized that the most frequent manifestations of PNS vasculitis are asymmetric, sensorimotor polyneuropathy [31–33]. Although any nerve can be affected, most patients have initial symptoms in the tibial or peroneal nerve [16,34]. Other studies found that sensory nerves are more sensitive for pathological changes than motor nerves and the sural nerve was the most frequent and the first nerve to be affected [16,35].

In our study there was no relationship between electrophysiological abnormalities and the drugs used. This finding suggests that neurological involvement is mostly related to the disease itself not to drug complications and these neurological changes can occur early in the disease course. So, one must be alert to these changes early in the course of the disease.

In this work we studied the clinical and subclinical neurological complications of common rheumatic disorders of childhood. As found by another study, the presence and the degree of nervous system impairment varied widely, depending on the diagnosis and course of the disorder [1]. Subclinical neurological abnormalities were common findings, so PNS and CNS evaluation is required for children with rheumatic CTDs, especially those with JSLE, for early diagnosis and estimation of peripheral and central neurological changes.

In conclusion, clinical neurological abnormalities are not common in JCTDs whereas subclinical neurological abnormalities affecting PNS, CNS and muscles are common findings. Subclinical myopathy is a common finding in JCTDs and mostly related to corticosteroid use. JSLE had the most common clinical (mononeuropathy) and subclinical neurologic abnormalities. In JIA, subclinical PNS involvement is less frequent and polyneuropathy is mostly demyelinating. A larger scale longitudinal study is recommended in future research.

Conflict of interest

None.

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