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ORIGINAL ARTICLE

Cross-sectional Study on Electrophysiological Evaluation of Neuropathy in Rheumatoid Arthritis

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Abstract

Introduction: Rheumatoid Arthritis (RA) is a chronic multisystem immune mediated disease. Rheumatoid associated neuropathy causes significant disability and adds to the economic burden. Aim of the study was to assess clinical determinants of peripheral neuropathy (diagnosed electro physiologically using nerve conduction studies) among patients with RA. Additionally, it was also aimed to study the various patterns of peripheral neuropathy in patients with RA. **Methods**: A cross-sectional study was conducted on 100 consecutive adult patients with RA between 01st February, 2020 to 02nd January, 2021 at medicine and neurology departments of SMS Medical College and Hospital, Jaipur, Rajasthan, India. Inclusion and exclusion criteria were followed and eligible patients after appropriate laboratory evaluation underwent nerve conduction studies. Statistical analysis was performed using student's unpaired t-test and Chi-square test for continuous and categorical variables respectively. **Results**: Mean age of the study population was 42.4±14.2 years with 88 females and 12 males. Mean duration of RA was 7.0±7.4 years. Nerve conduction studies detected neuropathy in 18 patients, of these only four patients were symptomatic with tingling, pins and needles sensation and numbness. Fourteen patients had subclinical neuropathy. Patients with neuropathy had significantly longer disease duration (p=0.0001), were older (p=0.014) with more joint deformities (p=0.0008). **Conclusion:** Subclinical neuropathy is not infrequent in RA patients. Those with advanced age, longer disease duration, higher Erythrocyte Sedimentation Rate (ESR), erosions and deformities should be assessed electro physiologically for neuropathy.

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Introduction

RA is a chronic multisystem immune-mediated disease characterised by symmetrical small and large joint polyarthritis. It affects approximately 0.5-1% of the adult population worldwide [1]. Akin to other autoimmune diseases RA occurs more frequently in females than males in 3:1 ratio [1]. Globally, in terms of years of life lived with disability RA was ranked as the 42nd highest contributor to disability amongst 291 conditions studied [2]. Extra-articular manifestations contributed to excess mortality in a community-based cohort of patients with RA [3]. The neurological manifestations of RA include peripheral neuropathy, cervical myelopathy due to atlantoaxial subluxation, entrapment neuropathy, mononeuritis multiplex and cerebral vasculitis [4,5]. Clinical assessment of neuropathy in RA is confounded by articular involvement which could cause wasting and painful restriction of joint mobility. Electrophysiological study of nerves to objectively assess neuropathy is a more reliable method of assessment therefore. Neuropathy is a significant contributor to the morbidity associated with RA [1].

Only few studies have focused on this aspect of RA [4-6], therefore this study was conducted with an aim to assess clinical determinants of peripheral neuropathy (diagnosed electro physiologically using nerve conduction studies) among patients with RA (diagnosed using 2010 American College of Rheumatology- European League against Rheumatism criteria) [7]. Additionally, it was also aimed to study the various patterns of peripheral neuropathy in patients with RA.

Materials and Methods

A cross-sectional study was conducted between February 2020 to January, 2021 in departments of neurology and medicine at tertiary care hospital from India. The sample size required were 91 cases with RA to verify an expected proportion of 39% patients with RA having peripheral neuropathy at 95% confidence interval and 80% power with 10% absolute error [7]. Worldwide authors have reported neuropathy in RA, most authors have reported neuropathy to be present in 20-40% of patients [8,9].

Inclusion Criteria

Hundred consecutive adults (>18 years) with RA, diagnosed according to 2010 American College of Rheumatology-European League against Rheumatism criteria (ACR-EULAR criteria [7]) presenting to medicine out-patient departments were enrolled.

Exclusion Criteria

Patients with the under mentioned conditions which are confounding factors for neuropathy were excluded: diabetes mellitus, impaired fasting and postprandial glucose or HbA1c $\geq$ 5.7%. For patients on steroids or with central obesity, dyslipidemia, additionally, oral glucose tolerance test was done to rule out impaired glucose tolerance and exclude patients with the same. Patients with hypothyroidism, uremia, history of chronic alcoholism, on or pervious use of neurotoxic drugs, hereditary, toxic, nutritional neuropathies, retropositive, Hepatitis B and Hepatitis C positive status, occupational exposure to neurotoxins, any other collagen vascular diseases, those with paraneoplastic neuropathy (including paraproteinemia associated neuropathy) and those too sick to consent were also excluded. Patients with low back ache, radicular pain, positive straight leg raising test with absent tibial H-reflex or prolonged peroneal and tibial F-waves suggestive of lumbar radiculopathy were also excluded. Written informed consent was obtained from patients at enrollment.

Detailed history including articular and extra-articular manifestations along with history of peripheral neuropathy was taken. Relevant past history, personal history (alcohol and smoking habits) and treatment history including disease modifying antirheumatic drug and steroid use was taken for all the 100 eligible patients.

Each patient was thoroughly examined and the particulars entered in proforma. Swollen (sw28), tender joint count (t28) and Visual Analog Scale (VAS) which features a horizontal line with the words NO PAIN on the left and WORST PAIN (explained to patient in local language) on the right were used. For VAS the patient made a mark on the line from 'No Pain to Worst Pain' to indicate the point on the spectrum that reflected how he or she was feeling. This mark was measured in mm, the range being 0 to 100 mm. These values along with Erythrocyte Sedimentation Rate (ESR) were entered in DAS 28 (Disease Activity Score 28) formula and the DAS 28 value was thus obtained [10].

All patients underwent complete blood count, random blood sugar, liver, renal function tests, rheumatoid factor, ESR, vitamin B12, Antinuclear antibody, anti-ds Deoxyribonucleic Acid (DNA), other autoantibodies as per requirement, thyroid function tests, Human Immunodeficiency Virus (HIV), Hepatitis B Surface Antigen (HBsAg) and Hepatitis C Virus (HCV) Enzyme Linked Immuno Sorbent Assay (ELISA), urine routine microscopy, X-ray of small joints of hands and chest X-ray.

All 100 patients then underwent nerve conduction study. Belly-tendon (active-reference) montage was used for doing nerve conduction maintaining ambient room temperature. All nerve conduction studies (motor orthrodromic and sensory antidromic) were done by two experienced neurophysicians independently. Motor responses were obtained in millivolts while sensory in microvolt range. Active recording electrode was placed on the center of the muscle belly (over the motor endplate) and the reference electrode was distally placed over the muscle tendon. The stimulator was placed over the nerve that supplies the muscle, with the cathode being placed closest to the recording electrode. The F response is a late motor response that occurs after the Compound Muscle Action Potential (CMAP) while the H reflex is a true reflex with sensory afferent, synapse and motor efferent.

Standard parameters measured were: latency, conduction velocity and amplitude of motor and sensory nerves with their F-waves and H reflexes. Median, ulnar, peroneal and tibial motor and sensory conduction parameters were recorded. Patient parameters were compared with standard accepted normal values for all the tested parameters [Table 1] [11].

Conduction velocity of $\leq$ 50 m/s in upper limbs and $\leq$ 40 m/s in lower limbs and distal latency more than upper limit of normal [Table 1] was considered as demyelination. Reduced compound motor action potential and sensory action potential with marginal reduction in velocity or latency prolongation was considered axonal affection [11].

Statistical Analysis

Data analysis was done using Statistical Package for the Social Sciences (SPSS) 20.0 statistical software. Quantitative data were summarised in the form of mean and Standard Deviation (SD) and the difference in means were analysed using student unpaired t-test. Categorical data was analysed using Chi-square test. Level of significance i.e., p-value <0.05 was kept as significant whereas <0.001 was kept as highly significant.

Table 1: Normal sensorimotor conduction parameters.

Nerve	Muscle recorded	Distal latency (ms)	Conduction velocity (m/s)	Amplitude (mV)	Distal distance (cm)
Median (M)	Abductor pollicis brevis	≤4.4	≥46	≥4	7
Ulnar (M)	Abductor digitiminimi	≤3.3	≥49	≥6	7
Median (S)	Digit 2	≤3.5	≥50	≥20	13
Ulnar (S)	Digit 2	≤3.1	≥50	≥17	11
Peroneal (M)	Extensor digitoru brevis	≤6.5	≥44	≥2	9
Tibial (M)	Abductor hallucis brevis	≤5.8	41	≥4	9
Superficial	Lateral ankle	≤4.4	40	≥6	14
Sural	Posterior ankle	≤4.4	40	≥6	14

[F-Latencies: Median ≤31ms Ulnar ≤32 ms Peroneal ≤56 msTibial≤56 ms, H-Latency: Tibial≤34 ms, M=Motor S-Sensory]

Results

The study comprised of 12 male and 88 female patients. Three patients (3%) had completed treatment (six-month ATT regimen) and were declared cured of pulmonary tuberculosis. Two females and four males (n=6) consumed three or lesser drinks per month while the rest of the study population were teetotallers. Eight females and five males (n=13) had a history of lesser than or equal to five pack years of smoking. One female and four male patients were taking alcohol and also smoked (n=5). Thirty-eight patients were on steroids and 85 on disease modifying drugs. Eighty-two patients had positive rheumatoid factor and 18 were negative for the same. Seventy-three patients (73%) had evidence of erosions in the X-ray of the small joints of the hands and 27 patients (27%) had a normal X-ray. Fifty-seven patients had a positive C-reactive protein. Most patients were in 30-39 years of age range, subgroup analysis however showed that most male patients with RA presented in the 50-59 years age group. Twenty-two patients were hypothyroid and six patients had antinuclear antibody positivity but were antids DNA negative. These six patients had no suggestion of systemic lupus erythematosus. No other autoantibodies were positive. Salient clinical characteristics are tabulated in [Table 2].

Table 2: Characteristics of study population

(n=100)

Paramter	Distal
Age (years)	42.4±14.2
Duration of RA (years)	7.0±7.4
Swollen joint count	9.3±10.2
Tender joint count	12.6±10.3
Visual analog scale	62.1±24.8
Disease activity score 28	5.7±1.9
Haemoglobin (g%)	11.4±1.6
Erythrocyte sedimentation rate (in mm/hr)	36.3±23.4

Eighteen patients showed evidence of neuropathy on electrophysiological testing. Ten (55.56%) had motor neuropathy, five (27.78%) had sensorimotor neuropathy and three patients (16.66%) had only sensory neuropathy. Sixteen out of 18 patients (88.9%) with nerve conduction studies proven neuropathy showed axonal neuropathy. Only one patient showed a demyelinating neuropathy (5.6%) and one patient showed features of both axonal and demyelinating neuropathy (5.6%).

Four out of 18 (22.22%) patients with electro-physiologically proven neuropathy complained of symptoms of neuropathy with the rest (14/18, 77.78%) of the patients having subclinical neuropathy. Tingling, pins and needles sensation and numbness was reported by symptomatic patients. Peroneal nerve was most commonly involved (in 14 patients) with median and ulnar being equally involved (in eight patients each). Sural nerve was involved in four patients and tibial nerve was the least affected in only two patients. Tibial H-latency was normal in all patients. Median F latency was mildly prolonged (33, 34 msec, respectively) in two patients with sensorimotor affection of median nerve. F latencies for all tested nerves except the patients previously mentioned were normal. Median neuropathy was noted in eight patients with entrapment neuropathy at wrist (carpal tunnel syndrome) in six patients.

Patterns of nerve involvement for all patients with neuropathy is shown in [Table 3] and mean values of latencies, amplitude and velocity for all tested nerves were recorded [Table 4]. Patients with neuropathy had significantly longer disease duration (p=0.0001), were older (p=0.014) with more joint deformities (p=0.0008) and had higher erythrocyte sedimentation rate (p=0.04) [Table 5].

Discussion

Nervous system involvement in RA can occur at the level of central nervous system, peripheral nerves, autonomic nervous system, muscle or neuromuscular junction, singly or in combination. Neuropathy complicating RA can be broadly divided into entrapment and non-entrapment neuropathy. The most common entrapment site is median nerve compression at the Carpal Tunnel Syndrome (CTS). Tarsal tunnel syndrome with compression of posterior tibial

Table 3: Patterns of neuropathy in all affected patients (n=18).

Patient number	Median	Ulnar	Personal	Tibial	Sural	Axonal/Demyelinating
1			M (B/L)			Ax
2	SM (B/L)					de
3			M (B/L)			Ax
4			M (B/L)			Ax
5		M (U/L)	M (B/L)			Ax
6			M (B/L)			Ax
7	M (U/L)	M (U/L)	M (B/L)			Ax
8	SM (B/L)	SM (B/L)	SM (B/L)	SM (B/L)	B/L	Ax
9			M (B/L)			Ax+De
10	SM (U/L)	SM (U/L)	SM (B/L)		U/L	Ax
11	SM (B/L)	SM (B/L)	SM (B/L)	SM (B/L)	B/L	Ax
12			SM (B/L)		U/L	Ax
13	S (UL)	S (UL)				Ax
14			M (B/L)			Ax
15			M (B/L)			Ax
16		S (B/L)				Ax
17	M (U/L)	M (U/L)	M (B/L)			Ax
18	S (B/L)					Ax

Ax: Axonal; De: Demyelinating; M: Motor; S: Sensory; SM: Sensorimotor; U/L: Unilateral; B/L: Bilateral

Table 4: Electrophysiological parameters of tested nerves.

Nerve	Motor (Mean±SD)			Sensory (Mean±SD)		
	Latency msec	CMAP mv	Velocity m/s	Latency Msec	SNAP µV	Velocity m/s
Median	4.13±0.40	3.83±0.56	49.33±4.02	3.36±0.42	19.16±3.09	53.33±5.13
Ulnar	3.10±0.31	8.06±3.86	50.66±2.85	3.07±0.37	15.66±2.40	53.88±4.58
Peroneal	6.13±0.32	1.95±0.43	43.77±2.30	4.12±0.29	6.15±0.81	41.99±2.60
Tibial	4.05±0.30	5.50±0.39	41.2±3.37	-------	-------	-------
Sural	-------	-------	-------	4.29±0.28	42.11±1.30	5.85±1.34

CMAP: Compound muscle action potential; SNAP: Sensory nerve action potential

nerve beneath the flexor retinaculum below the medial malleolus of the ankle is also relatively common. The neuropathic pain complicates the pre-existing arthritic pain and is often overlooked [12]. The other involved sites are ulnar nerve at the elbow and anterior tibial nerve at the fibular head. Six patients in present study had median entrapment neuropathy. At other sites entrapment was not recorded in present study. The non-entrapment neuropathies are sensory neuropathy, sensorimotor neuropathy, mononeuritis multiplex and motor neuropathy. Necrotising vasculitis with or without systemic vasculitis has been thought to be chiefly responsible for the different patterns of non-compressive neuropathies in RA [13]. Long standing RA, male sex, smoking, rheumatoid nodules and in particular Human Leukocyte Antigen (HLA) class I and class II genotypes, hypocomplimentemia and circulating cryoglobulins have been associated with an increased risk of vasculitis [1]. The two common clinical patterns are symmetric distal sensory neuropathy and mononeuritis multiplex (sensorimotor neuropathy) [9].

The prevalence of neuropathy varies widely in different studies. Agarwal V et al., studied 100 patients of RA and detected peripheral neuropathy in 60%, while Aneja R et al., reported electrophysiological evidence of neuropathy in 37.87% of RA patients [5,9]. Biswas M et al., examined the nerve conduction in 74 patients with RA of at least two years duration [6]. Peripheral neuropathy was detected in 39.19% i.e., 29 out of 74 patients. In contrast to these studies present

Table 5: Comparison of patients with neuropathy vs without neuropathy

Parameter	Neuropathy (18) Mean±SD or counts of patients	Non-neuropathy (82) Mean±SD or counts of patients	p-value
Mean age (years)	49.9±13.5	40.9±14	0.014
Females	17/18 (94%)	71/82 (87%)	0.35
Disease duration (years)	13.0±6.4	5.7±6.9	0.0001
RA factor positive (n=82)	15 (83.3%)	67 (81.7%)	>0.05
Erosions of joint X-rays (n=73)	18 (100%)	55 (67%)	0.07
DMARD use (n=85)	16 (89%)	69 (84%)	0.60
ESR (mm/hr)	46.3±25.6	34.1±22.1	0.04
CRP>10 mg/L (n=57)	10 (57%)	47 (56%)	0.89
Deformities present (n=33)	12 (67%)	21 (26%)	0.008
Disease activity score	5.9±2	5.6±1.9	0.62
Swollen joint count	9.1±9.6	9.3±10.4	0.94
Tender joint count	11.9±9.7	12.8±10.4	0.74
Visual analog scale	65±26.4	61.5±24.5	0.58

study detected neuropathy in fewer (18%) patients. This wide variation in results could be due to differences in inclusion and exclusion criteria, patients with varying disease duration as well as method of diagnosis of neuropathy. The lesser incidence in present study could also be due to declining incidence of vasculitis in recent times with availability of more effective disease modifying drugs.

Only 22.22% of the patients had clinical neuropathy with the rest being subclinical neuropathy (77.78%). All the symptomatic patients presented with tingling, pins and needles sensation, numbness with no motor weakness. Studies by Nadkar MY et al., and Aneja R et al., also showed majority of patients had subclinical neuropathy with no overt symptoms, with sensory symptoms exceeding motor weakness [4,9]. In present study, 55.56% (10 out of 18) of the electro-physiologically proven neuropathies were of motor type, 27.78% (5 out of 18) were of sensorimotor type and 16.66% (3 out of 18) were sensory type. The frequency of sensorimotor neuropathy was higher than isolated sensory or motor neuropathy in studies by Nadkar MY et al., and Aneja R et al., [4,9]. Besides vasculitis, an antibody mediated attack on the peripheral nervous system antigens (gangliosides) is thought to be responsible for the neuropathy in RA [14]. Keeping in view the high association of anti-ganglioside antibodies seen in various clinical motor neuropathies, study of this antibody as an alternative cause of motor neuropathy in RA is warranted and could be a possible explanation of the higher prevalence of motor neuropathy seen in present study as compared to others.

Axonal neuropathy was most common at 88.9% (16 out of 18 patients) followed by axonal plus demyelinating and least common was demyelinating. This is akin to electrophysiological pattern of neuropathy in various other studies [5,6]. Similar to the study by Aktekin L et al., present study also noted predominantly peroneal nerve involvement [15]. This is likely due to the axonal dying back neuropathy noted in RA.

In the present study it was noted that older patients with longer disease duration to be more significantly associated with neuropathy. Older age is a well-known risk factor for polyneuropathy. The correlation of duration of disease with neuropathy can be explained on the basis of occurrence of vasculitis which occurs in long standing disease and is the foremost cause of neuropathy [1]. No gender predilection was noted in development of neuropathy in the present study similar to Biswas M et al., [6]. Although a few studies have shown neuropathy to be more common in males, it was not seen in this study [9]. This could be due to higher number of female than male patients in present study. Seropositivity of RA factor (82%) was similar to other studies and like most studies this was not significantly associated with development of neuropathy [1,5]. Present study did not find any relation between use of Disease-modifying antirheumatic drugs (DMARDs) and development of neuropathy similar to other studies [6]. Similar to present study, Nadkar MY et al., found ESR to be significantly associated with neuropathy [4]. Raised CRP and neuropathy had no association in present study.

Significant association between deformities and neuropathy was noted in present study similar to Bharadwaj A and Haroon N [16]. As patients with neuropathy in present study have longer duration of disease, it was naturally associated with higher percentage of deformities. Vasculitis, the primary cause of peripheral neuropathy is also predominantly seen

in patients with severe deforming arthritis and extra-articular manifestations. This explains the greater prevalence of deformities in patients with neuropathies. The mean swollen and tender joint count for patients with neuropathy was comparable to those without neuropathy. Bharadwaj A et al., also reported that the clinical profile of patients with and without extra-articular manifestations, assessed with swollen, tender joint count and early morning stiffness did not vary within the groups.

Limitations

Firstly, most of the patients with neuropathy had isolated motor neuropathy. This is unlike other studies wherein sensorimotor neuropathy was the dominant type. This could be due to a relatively small sample size and single centre study. Secondly, longitudinal follow-up studies to assess the progression of neuropathy could not be done. Thirdly, nerve biopsy was not done, this would have given a neuro-pathological correlation. Fourthly, characterisation of the duration and dose of DMARD should have been done to eliminate any contamination caused by drug induced neuropathy. Levamisole is associated with development of neuropathy in RA patients, this drug was very sparingly used in a few patients in this study.

Conclusions

Subclinical neuropathy is not infrequent in RA patients and this contributes to the disability associated with RA. Those with advanced age, longer disease duration, deformities and high ESR should be assessed electro-physiologically for neuropathy. This would ensure optimisation of treatment and reduce the disability associated with neuropathy.

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References

1. Kasper D, Fauci A, Hauser S, Longo D, Jameson J, Loscalzo J. Harrison's principles of internal medicine, 19e. New York, NY, USA:: Mcgraw-hill; 2015.
2. Hoy D, March L, Woolf A, Blyth F, Brooks P, Smith E, Vos T, Barendregt J, Blore J, Murray C, Burstein R. The global burden of neck pain: estimates from the global burden of disease 2010 study. Annals of the rheumatic diseases. 2014 Jul 1;73(7):1309-15.
3. Turesson C, O'Fallon WM, Crowson CS, Gabriel SE, Matteson EL. Occurrence of extraarticular disease manifestations is associated with excess mortality in a community based cohort of patients with rheumatoid arthritis. The Journal of rheumatology. 2002 Jan 1;29(1):62-7.
4. Nadkar MY, Agarwal R, Samant RS, Chhugani SJ, Idgunji SS, Iyer S, Borges NE. Neuropathy in rheumatoid arthritis. The Journal of the Association of Physicians of India. 2001 Feb 1;49:217-20.
5. Agarwal V, Singh R, Chauhan S, Tahlan A, Ahuja CK, Goel D, Pal L. A clinical, electrophysiological, and pathological study of neuropathy in rheumatoid arthritis. Clinical rheumatology. 2008 Jul;27(7):841-4.
6. Biswas M, Chatterjee A, Ghosh SK, Dasgupta S, Ghosh K, Ganguly PK. Prevalence, types, clinical associations, and determinants of peripheral neuropathy in rheumatoid patients. Annals of Indian Academy of Neurology. 2011 Jul;14(3):194.
7. Kay J, Upchurch KS. ACR/EULAR 2010 rheumatoid arthritis classification criteria. Rheumatology (Oxford). 2012;51(Suppl 6):vi5-9. doi: 10.1093/rheumatology/kes279.
8. Albani G, Ravaglia S, Cavagna L, Caporali R, Montecucco C, Mauro A. Clinical and electrophysiological evaluation of peripheral neuropathy in rheumatoid arthritis. J PeripherNerv Syst. 2006;11:174-75.
9. Aneja R, Singh MB, Shankar S, Dhir V, Grover R, Gupta R, Kumar A. Prevalence of peripheral neuropathy in patients with newly diagnosed rheumatoid arthritis. Indian Journal of Rheumatology. 2007 Jun 1;2(2):47-50.
10. Fransen J, Stucki G, van Riel PL. Rheumatoid arthritis measures: disease activity score (DAS), Disease activity score28 (DAS28), Rapid assessment of disease activity in rheumatology (RADAR), and Rheumatoid arthritis disease activity index (RADAI). Arthritis Care & Research: Official Journal of the American College of Rheumatology. 2003 Oct 15;49(S5):S214-24.
11. Preston DC, Shapiro BE. Electromyography and Neuromuscular disorders. 2nd ed. Elsevier, 2005; Appendix. 663-665.
12. Agrawal V, Usha, Goel SC, Gupta RM. A probable explanation for mild extra-articular manifestations in Indian patients of rheumatoid arthritis: A preliminary study. Indian J PatholMicrobiol. 1998;41(3):297-301.
13. Puéchal X, Said G, Hilliquin P, Coste J, JobDeslandre C, Lacroix C, Menkès CJ. Peripheral neuropathy with necrotizing vasculitis in rheumatoid arthritis. Arthritis & Rheumatism: Official Journal of the American College of Rheumatology. 1995 Nov;38(11):1618-29.
14. Salih AM, Nixon NB, Gagan RM, Heath P, Hawkins CP, Dawes PT, Mattey DL. Anti-ganglioside antibodies in patients with rheumatoid arthritis complicated by peripheral neuropathy. Rheumatology. 1996 Aug 1;35(8):725-31.
15. Aktekin LA, Gözlükaya H, Bodur H, Borman P, Köz Ö. Peripheral neuropathy in rheumatoid arthritis patients: an electroneurophysiological study. Archives of Rheumatology. 2009;24(2):062-6.
16. Bharadwaj A, Haroon N. Interstitial lung disease and neuropathy as predominant extra-articular manifestations in patients with rheumatoid arthritis: a prospective study. Medical science monitor: international medical journal of experimental and clinical research. 2005;11(10):CR498-502.