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CASE SERIES

Sporadic inclusion body myositis – single center case series of 8 patients from a fifteen-year period

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Summary

Objective: Sporadic inclusion body myositis (IBM) is a slowly progressive inflammatory myopathy. Clinical presentation comprises asymmetric distal and proximal limb weakness and dysphagia. Muscle biopsy showing rimmed vacuoles are the main diagnostic indicator.

Method: We showed 8 patients with an IBM at the Neurology Clinic of the University Clinical Center Serbia in the period 2009-2024. We analyzed medical records, focusing on the following characteristics: sociodemographic data, age and presenting symptoms at disease onset, comorbidities, findings from neurological examinations, IBM functional rating scores, and results from laboratory and diagnostic procedures.

Results: The average age of years at the onset of the disease was 57.7±0.4 years. The first signs of disease were difficulty walking, dysphagia, hand weakness and eyelid ptosis. The average IBMFRS was 28.7±7.8. Three patients had clinical and ENG signs for polyneuropathy. In four patients, MRI revealed muscle degenerative changes consistent with grade 2b. Muscle biopsy was performed in seven patients and they fulfilled the criteria for clinically defined IBM. Five patients were treated with IVIG with minimal and short-term improvement.

Conclusion: Clinical examination and muscle biopsy are essential for establishing a diagnosis of IBM. Early diagnosis and early administration of the right therapy would make the greatest contribution to slowing the progression of the disease.

Key words: sporadic inclusion body myositis, case series, muscle biopsy, muscle MRI

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INTRODUCTION

Sporadic inclusion body myositis (IBM) is the most common inflammatory myopathy which begins over 45 years of age. The 272nd ENMC International Workshop, held in June 2023, marked a significant milestone in the understanding and management of IBM. Building upon the 2013 ENMC diagnostic criteria, the workshop introduced several key updates reflecting advancements over the past decade. The updated criteria aim to enhance diagnostic accuracy by incorporating recent insights into IBM's pathogenesis and clinical presentation. Emphasis is placed on early recognition, especially in atypical cases and younger patients, acknowledging the broader spectrum of disease manifestations. The criteria now integrate novel diagnostic tools, including muscle imaging techniques like MRI and ultrasound, and serological testing for cytosolic 5'-nucleotidase-1A (cN1A) antibodies (1). The prevalence of IBM is 24.8/1 000 000 (2). Studies have shown an association of IBM with the HLA-DRB1*03, DRB*03:01, DRB1*01, DRB1*01:01, DRB1*15:02, B*08, and the DQB1*02 allele (3). The IBM pathogenesis is still undetermined, and probably multifactorial, but there are two possible hypotheses: the autoimmune hypothesis and the degenerative hypothesis (2,4).

The diagnostic criteria for IBM are: common presentation (age ≥ 45 years at symptom onset, ≥12-month history of progressive weakness, CK ≤ 15x ULN) with common muscle IBM involvement pattern at presentation (often asymmetric and accompanied by dysphagia): deep finger flexor (FF) weakness and/or knee extensor (KE), and muscle biopsy findings (mandatory: inflammation consisting of endomysial lymphocytes surrounding non-necrotic muscle fibers (with or without invasion)), or supportive (1. rimmed vacuoles *and/or* cytoplasmic protein aggregates; 2. mitochondrial abnormalities (COX-SDH+ fibers > age-related); 3. anti-cN1a autoantibody positive; 4. typical muscle MRI appearance *and/or* typical muscle ultrasound pattern). Diagnosis of IBM is confirmed when there is: common presentation with FF and KE weakness, and mandatory investigation finding (1).

Antibodies targeting cytosolic 5'-nucleotidase 1A (cN-1A) are currently the only serum-based diagnostic marker for IBM. Although enzyme-linked immunosorbent assay (ELISA) is the most widely used method for their detection, the test's sensitivity remains relatively low (30% to 50%). In contrast, the specificity of cN-1A antibodies is generally high—over 90%. However, this specificity decreases significantly in patients with other connective tissue disorders, including systemic lupus erythematosus, Sjögren's syndrome, and dermatomyositis, where up to one-third may test positive for cN-1A antibodies despite not having IBM. Therefore, cN-1A antibody results should be interpreted with caution and always considered in the context of the patient's clinical presentation (5).

To-date literature underlies that most of the investigated therapeutic strategies were insufficiently successful and that further randomized controlled trials are needed. However, there are conflicting results for some drugs (intravenous immunoglobulin (IVIG), arimoclomol, follistatin, canakinumab, bimagrumab,…). As a symptomatic approach, botulinum toxin injections in upper esophageal sphincter can improve dysphagia (6,7). Therefore, IBM surely represents a great economic burden of the health system (8).

Our study provides a clinical and pathological workup of a single center group of 8 patients with IBM from a fifteen-year period.

MATERIAL AND METHOD

We performed a retrospective analysis of all hospitalized patients at the Department of Neuromuscular Diseases of the Neurology Clinic of the University Clinical Center Serbia (UCCS), Belgrade in the period 2009-2024. with a diagnosis of myositis. We identified 8 patients with an IBM diagnosis. This retrospective study has been conducted in accordance with all ethical principles of the Declaration of Helsinki and in accordance and approval with all national and institutional ethical standards. All patients had negative family history.

We analyzed medical records and following characteristics: sociodemographic (age and gender), age and symptoms/signs at onset of disease, co-morbidities, neurological examination, fulfillment of the 2024. diagnostic criteria for IBM (1), analysis of all other laboratory and other supplementary available diagnostic procedures performed during hospitalization (muscle CT, muscle MR, ENG, EMG, muscle biopsy). After the biopsy of the muscles, the sample was analyzed by microscopy. We calculated an IBM functional rating score (IBMFRS) and Barthel Index based on functional disability. IBMFRS has a maximum value of 40 indicating that the patient is without functional disability in the 10 domains examined by this scale (9).

Depending on the type of variables and the normality of the distribution, data will be presented as n (%), mean ± standard deviation, or median (range).

RESULTS

The average age of years at the onset of the disease was 57.7±10.4 years, and the average age at the time of first hospitalization was 63.2±8.17 years. The average duration of disease until the first hospitalization and definite diagnosis at the Neurology Clinic UCCS was 6.75 (min: 0, max: 18 years). All patients had a characteristic distribution of hypotrophy and muscle weakness of varying degrees (weakness of the flexors of the hands, hypotrophy and weakness of the m. quadriceps, weakness of the muscles in the anterior compartment of the lower legs) **(table 1)**.

Table 1. Characteristics of patients with sporadic inclusion body myositis (sIBM)

	Number of patients							
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**
Sociodemographic characteristics								
Gender	M[1]	M	M	F[2]	F	M	F	F
Age	59	66	70	70	51	64	73	71
Characteristics of diseases								
Comorbidities	HTA[3] Hemorrhoids Hepatomegaly Hiatus hernia HLD[4]	HTA	HTA AF[5] HLD Lichen chronicus	Right eye cataract HLD	No	HTA AAA[11]	Tachy-cardia Discus hernia	AF HTA
Symptoms on IBM onset	Walking	Walking	Dysphagia	Walking	Ptosis (right)	Hands	Walking	Walking
Duration of disease (y)	18	4	3	2	6	2	13	15
Dysphagia	Yes	No	Yes	Yes	Yes	No	No	Yes
Diagnostic criteria for clinically defined IBM								
I	41	62	67	68	45	62	52	65
II (years)	18	4	3	2	6	2	13	15
III	641	747	599	1145	161	831	500	800
IV	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
V	Yes	NA[6]	Yes	Yes	Yes	Yes	Yes	Yes
Other clinical and diagnostic characteristics								
LDH[7] (220-460 IU/L)	583	716	703	580	374	439	482	NA
Asymmetry	Right	Right	Left	Right	No	No	No	No
Myopathic EMG[8]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Neuropathy on ENG[9]	No	Yes	Yes	No	No	No	Yes	No
Radiculopathy on ENG	No	No	Yes	No	No	No	Yes	No
IBMFRS	28	37	21	32	14	29	34	35
Barthel index	60	80	60	65	25	88	90	90
Mobility	Amb[10]	Amb	Amb	Amb	Wheelchair	Amb	Amb	Amb
Therapy								
Therapy during disease	No	No	IVIG[12]	IVIG	IVIG	IVIG	No	IVIG

I- Age of onset > 45 years; **II**- Duration > 12 month; **III**- Creatine kinase < 15x normal (0-200 U/L); **IV**- With common IBM muscle involvement pattern at presentation: Deep finger flexor (FF) weakness AND/OR Knee extensor (KE) weakness; **V**- Inflammation consisting of endomysial lymphocytes surrounding non-necrotic muscle fibers (with or without invasion)
[1]Male [2]Female [3]Hypertension [4]Hyperlipidaemia [5]Atrial fibrilation [6]Not available [7]Lactate dechydrogenase [8]Electromyography [9]Electroneurography [10]Ambulatory[11] Abdominal aortic aneurysm [12] Intravenous immunoglobulin

Muscle biopsy was performed in 7/8 (87.5%) patients and the diagnosis of IBM was confirmed. The eighth patient had not undergone a muscle biopsy and therefore met the criteria for a probable diagnosis of IBM. The creatine kinase (CK) level was 694 U/L (min 161 U/L, max 1145 U/L) and LDH level was 553.9±129.6 U/L. Anti-cN1a autoantibodies were not performed on any patient. At the time of hospitalization, the average IBMFRS was 28.7±7.8 and Barthel index was 69.7±22.3.

Five patients were treated with IVIG at a dose of 0.4 g/kg daily for five days, followed by a booster dose of 0.4 g/kg every 6-8 weeks for at least 6 months. Minimal

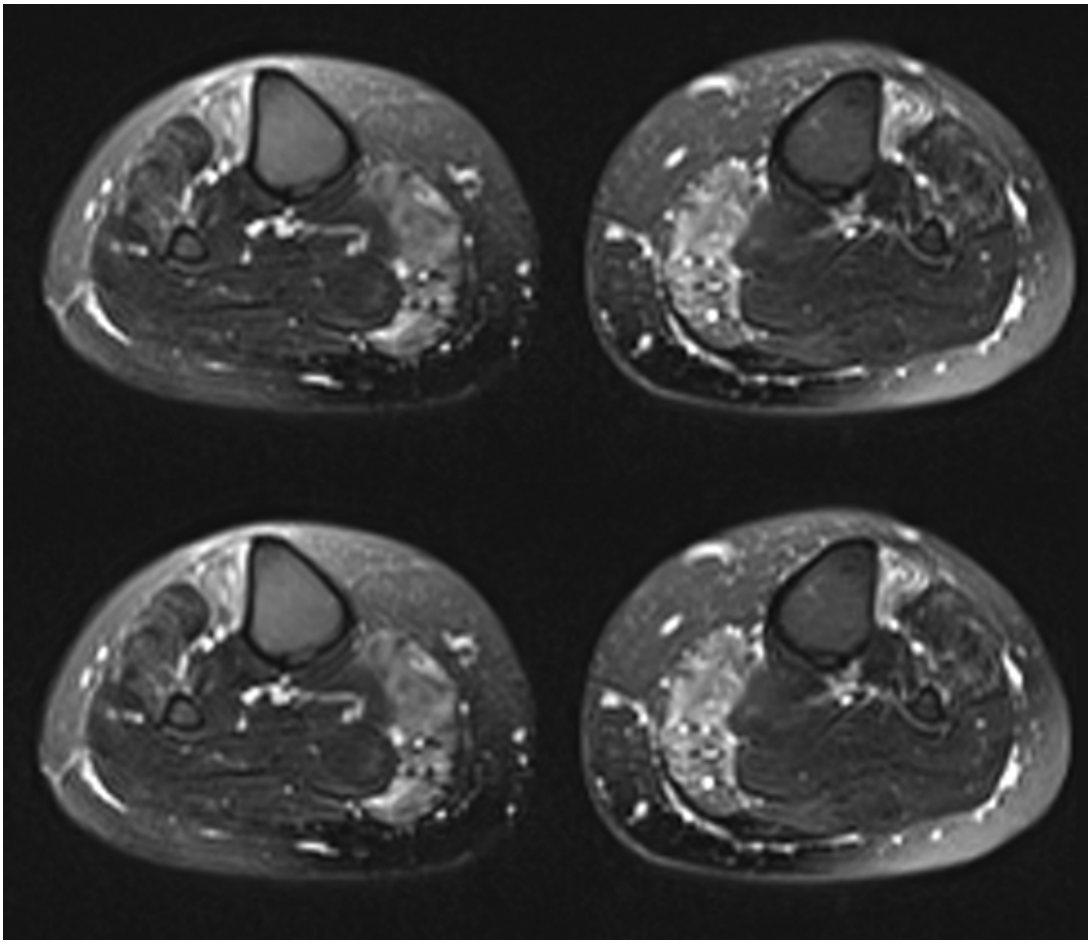


Figure 1. Symmetrical diffuse muscle hypotrophy of all compartments of both upper legs and lower legs, with diffuse edema and fatty degeneration in the distal third of the vastus medialis and lateralis m. gastrocnemius medialis, m. tibialis anterior.

and short-term improvement was noted. These and other clinical and laboratory characteristics were shown in **Table 1**.

Serum protein immunoelectrophoresis in patient 1 revealed the presence of polyclonal IgG antibodies, while urine protein immunoelectrophoresis results were normal. On the abdomen ultrasound, the diameter of the liver was larger (190 mm) than normal. CT of the proximal third of the thighs showed atrophy and fat infiltration and CT of the lower legs were normal. Patient number 2 had dysesthesias on his legs in the form of short socks and hands in the form of short gloves, while electroneurography (ENG) showed sensory and motor axonal and demyelinating polyneuropathy on upper extremities (UE) and lower extremities (LE), which was more expressed on LE. The etiology of motor-axonal polyneuropathy remained idiopathic. For patients 4,6,7, and 8 MRI of the proximal third of the thighs and the lower legs showed muscle degenerative changes and thighs muscle edema **(Figure 1)** which was consistent with IBM diagnosis.

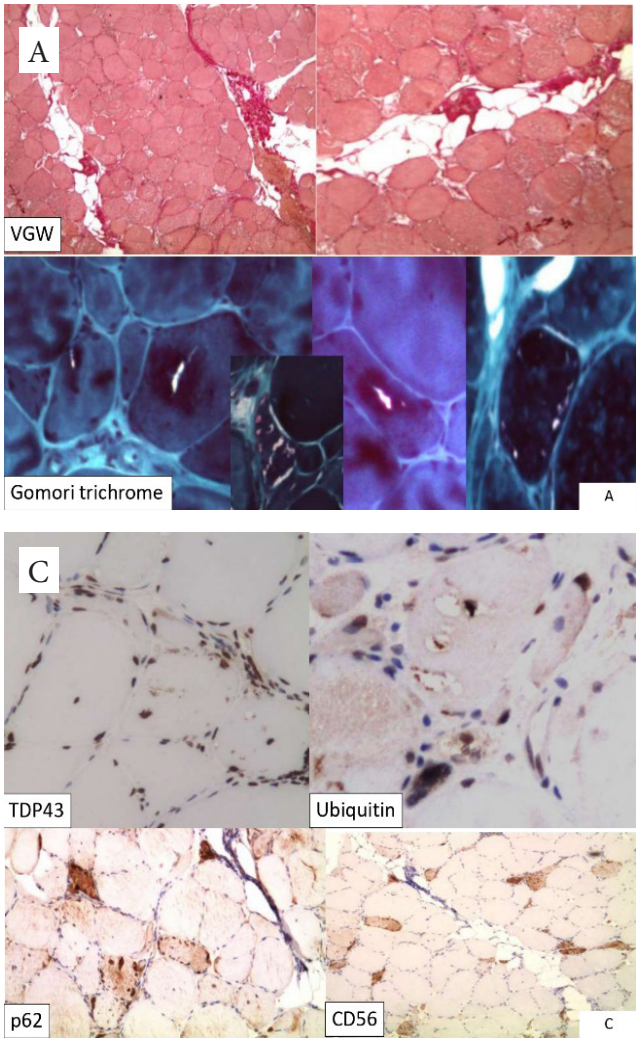
In the majority of patients (7/8), pathohistological examination showed findings characteristic of IBM and in all of them the pathohistological criteria for IBM were fulfilled **(Figure 2A, 2B, 2C)**.

DISCUSSION

We showed 8 patients with IBM and 7 patients had typical muscle biopsy findings for IBM. The course of the disease was atypically very slow but still progressive in three patients. The onset of disease in one of our patients in the form of ptosis was a very rare clinical manifestation.

In the largest percentage of patients, the IBM begins with difficulty walking, mostly due to the weakness of m. quadriceps. In five patients the first sign was difficulty walking, and in two patients the first problem was dysphagia. Dysphagia was a common presenting symptom being more frequent in women than men and was during the disease course reported in 74% of men and 84% of women (10). In our patient number 5, the first sign was right eyelid ptosis that lasted a month. In this patient, IBM started 6 years before hospitalization, but she has the highest functional disability (IBMFRS 14). Extraocular muscle weakness and ptosis is a very rare initial manifestation of IBM described only as a case report (11).

The average duration of the disease until definitive diagnosis is 4.6-5.8 years (1). In 5/8 (62.5) of our patients the diagnosis was made within 6 years, while in patient 1, a total of 18 years has passed from the onset of the disease. The very slow progressive course of the disease contributed significantly to this delay. Change in percentage of



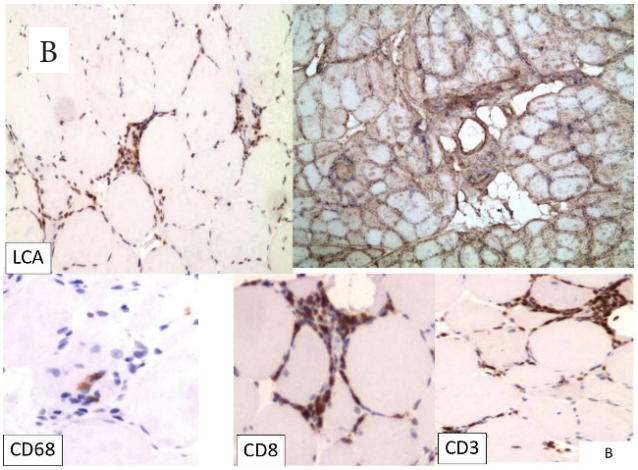


Figure 2. A. VGW: Proliferation of connective and fatty tissue is moderate, more pronounced interfascicularly. PAS: Glycogen content is preserved. Gomori trichrome: The distribution and density of mitochondria is grossly disturbed. Irregular to oval vacuoles are clearly visible and most of them contain material that is colored red (according to the "rimmed" vacuole type). Significant cytoplasmic bodies can also be observed in certain fibers.
B. A focal inflammatory infiltrate of the mononuclear type (LCA+) was observed with the presence of macrophages (CD68+) and a clearly expressed phenomenon of myophagocytosis. The expression of the host compatibility complex (MHC I) is disturbed with increased intensity both in the inflammation zone and with expression on the membrane and in the cytoplasm. The immune profile of lymphocytes is T type (dominantly CD8+ and CD3+).
C. The presence of positive fibers, sarcoplasmically granulated in the form of protein aggregates of the autophagic marker p62 and the positivity of the protein aggregating marker TDP-43, as well as Ubiquitin was observed. Angular fibers and fibers in active denervation express NCAM (CD56+) on the membrane and in the cytoplasm.

IBMFRS score over time yielded an average decline of 6.3% per year, with steeper decline in the initial years. Older age of onset was associated with a more rapid IBMFRS decline (12).

Up to 25% of IBM patients may have normal serum CK level (13). CK level is not associated with muscle strength, age, age at onset, and duration of disease (14). Our patient number 5 with the most severe deficit had a CK at the reference values. LDH was elevated in 5/8 (62.5%) of our patients. The recent case series showed that LDH levels were slightly elevated, periodically very elevated and that LDH levels did not correlate with CK levels (15). Also, serum markers did not have a statistically significant correlation with any of the clinical measures (16).

Some studies have shown an increased incidence of hypertension (HTA), hyperlipidemia (HLD) and myocardial infarction in the patients with IBM (17). In our sample, 6/8 (75%) patients had some of these diagnoses **(Table 1)**. On the other hand, more recent research has not confirmed this, but has found a higher incidence of peripheral neuropathy, Sjogren's syndrome and hematologic malignancies. (18) Three (37.5%) patients had clinical and ENG signs for sensorimotor axonal and demyelinating peripheral neuropathy. Even after all the diagnostic tests were done, the polyneuropathy remained idiopathic.

Although IBM is a slowly progressive disease that leads to wheelchairs for a mean time of 12 years (10), some patients had minimal progress after 12 years of disease (18). Despite having lived with IBM for 18, 13, and 15 years respectively, patients 1, 7, and 8 remain ambulatory, though with marked difficulty. IBM was associated with increased mortality risk compared with population controls, with hazard ratio per 1-year increase of 2.69 [1.74, 4.15] ($p < 0.0001$) (18).

Three placebo-controlled studies were performed with IVIG in patients with IBM. Although there was an improvement in the study groups in comparison with the control group, the differences were not statistically significant. Owing to its limited quality, this study was excluded from meta-analyses. Better-designed studies are needed (6,7,20). Our four patients received the IVIG recommended dose regimen, but the effects were short-lived (about three months) and therapy had the greatest positive effect on dysphagia. However, based on the most recent guidelines, IVIG is no longer considered an appropriate treatment for IBM, and it is no longer used for this purpose in Serbia.

Limitations

This study is limited by the small sample size which restricts the generalizability. The retrospective design may also introduce bias due to incomplete or inconsistent medical records. Not all patients underwent the same diagnostic procedures leading to potential variability in diagnostic certainty. Additionally, treatment outcomes, particularly with IVIG, were not assessed using standardized follow-up protocols, limiting conclusions regarding therapeutic efficacy. Finally, the absence of a control group prevents comparison with other myopathies or treatment modalities.

CONCLUSION

The IBM is a slow progressive disease of unknown etiology and unclear pathogenesis, with no effective therapy. Clinical examination and muscle biopsy are paramount in making the diagnosis. Early diagnosis and the detection of disease mechanisms in these patients are very significant. Early administration of the right therapy would make the greatest contribution to slowing the progression of the disease.

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Conflict of Interest Statement: No conflict of interest to report.

Authors' Contributions:

Conception and design: DA, IB, VRS; Data collection: DA, IB, VRS; Statistical analysis: DA, IB, VRS; Writing the article: DA, IB, VRS; Critical revision of the article: DA, IB, IK, SG, IB, VRS; Final approval of the article: DA, IB, IK, SG, IB, VRS

Ethical Approval: This retrospective study has been conducted in accordance with all ethical principles of the Declaration of Helsinki and in accordance and approval with all national and institutional ethical standards.

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SPORADIČNI MIOZITIS SA INKLUZIVNIM TELAŠCIMA – SERIJA SLUČAJEVA 8 PACIJENATA IZ JEDNOG CENTRA IZ PERIODA OD PETNAEST GODINA

Dejan Aleksić[1], Ivo Božović[2], Ivana Kezić[3], Sanja Gluščević[4], Ivana Basta[5], Vidosava Rakočević-Stojanović[5]

Sažetak

Uvod: Sporadični miozitis sa inkluzionim telima (IBM) je sporo progresivna inflamatorna miopatija. Klinička slika se sastoji od asimetrične slabosti distalnih i proksimalnih mišića ekstremiteta i disfagije. Biopsija mišića koja pokazuje "rimmed" vakuole je glavni dijagnostički indikator.

Metod: Prikazali smo 8 pacijenata sa IBM na Klinici za neurologiju Univerzitetskog kliničkog centra Srbije u periodu 2009-2024. Analizirali smo medicinsku dokumentaciju i sledeće karakteristike: sociodemografske podatke, uzrast i znakove bolesti, komorbiditete, neurološki nalaz, IBMFRS, rezultate laboratorijskih i dijagnostičkih procedura.

Rezultati: Prosečna starost godina na početku bolesti bila je 57,7±0,4 godine. Prvi znaci bolesti bili su otežano hodanje, disfagija, slabost ruku i ptoza očnih kapaka. Prosečan IBMFRS bio je 28,7±7,8. Tri pacijenta su imala kliničke i ENG znakove polineuropatije. Kod četiri pacijenta MR je pokazala degenerativne promene mišića stepena 2b. Biopsija mišića urađena je kod sedam pacijenata i oni su ispunjavali kriterijume za klinički definitivan IBM. Pet pacijenata je lečeno IVIG sa minimalnim i kratkotrajnim poboljšanjem.

Zaključak: Klinički pregled i biopsija mišića su najvažniji u postavljanju dijagnoze IBM. Rana dijagnoza i rana primena prave terapije dali bi najveći doprinos usporavanju progresije bolesti.

Ključne reči: sporadični miozitis sa inkluzionim telima, serija slučajeva, biosija mišića, MR mišića

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