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Diagnosis problems in patients with chronic teraparsec

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Abstract

Tetraparesis is a condition of motor weakness of the four extremities that can indicate a serious neurological disorder. The case reported a 27-year-old woman with progressive weakness in her shoulder and thigh muscles for five months. The purpose of this study is to establish the comparative diagnosis of tetraparesis and determine its etiology through clinical, laboratory, radiology, and histopathological approaches. The research method used a case study approach with comprehensive examinations including electromyography (EMG), Magnetic Resonance Imaging (MRI), myopathy serology profile, and muscle biopsy. The results of the examination showed an increase in muscle enzymes (SGOT, SGPT, creatinine kinase), myositis-specific antibodies (NXP2 and SRP positive), and endomisal lymphocyte infiltration on muscle biopsy that supported the diagnosis of Idiopathic Inflammatory Myopathy subtype polymyositis. Patients showed significant clinical improvement after intravenous methylprednisolone therapy of 500 mg for three days and cyclophosphamide 500 mg of single dose. The discussion emphasized the importance of differentiating the etiology of tetraparesis between central, peripheral, and myopathic lesions to determine the appropriate management. In conclusion, early diagnosis and aggressive immunomodulatory therapy play an important role in preventing permanent disability as well as improving the prognosis of patients with polymyositis.

Keywords: Tetraparesis, Idiopathic inflammatory myopathy, Polimiositis, Diagnosis banding

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Introduction

Tetraparesis is defined as motor weakness involving all four extremities with reduced muscle strength but not completely disappeared, a clinical manifestation that indicates the presence of a serious neurological disorder (Oliveira et al., 2021). This condition requires rapid identification and immediate treatment to prevent significant morbidity and mortality (Oliveira et al., 2021). A systematic diagnosis approach based on anatomical localization including the central nervous system (cerebrum, brainstem, spinal medulla), peripheral nervous system, neuromuscular connections, and muscles become crucial in determining etiology and directing appropriate therapy (Oliveira et al., 2021).

In cerebrum lesions, bilateral stroke of the Anterior Cerebral Artery (ACA) is a very rare condition, recorded in only 2 out of 1,490 stroke cases in the Lausanne Stroke Registry (0.13%), while ACA infarction generally accounts for 0.3–4.4% of all ischemic strokes (Bogousslavsky & Regli, 1990; Gacs et al., 1983). Typical clinical manifestations are paraparesis or tetraparesis with impaired consciousness, akinetic mutism, and frontal dysfunction (Hedna et al., 2015). At the brainstem level, locked-in syndrome has a very low prevalence (about 0.07 per 100,000 population) with 67% of cases due to vascular etiology, mainly basilar artery occlusion, and mortality reaching 60% based on a review of 139 cases (Patterson & Grabois, 1986; Smith & Delargy, 2005). Central pontine myelinolysis, which can lead to locked-in syndrome, was found in 0.5% of postmortem brains and in 29% of post-liver transplant patients, generally associated with over-rapid correction of hyponatremia (Messert et al., 1979). Overall, strokes account for 10–15% of all stroke cases, with pontine infarction being the most common (60% of all brainstem infarctions) (Al-Shaikh et al., 2023).

At the spinal cord level, spinal cord infarction has an incidence of 1.5–3.1 per 100,000 person-years and accounts for 4.4% of spinal cord injuries as well as 1.2% of all strokes (Zalewski et al., 2017). Transverse myelitis has an incidence of 1–8 cases per million population per year (0.1–0.8 per 100,000) with a prevalence of 4.6–7.9 per 100,000 population (Berman et al., 1981; Bhat et al., 2010). Cervical spondylotic myelopathy, as the most common spinal cord disorder in the geriatric population, has an incidence of 4.04 per 100,000 person-years and a prevalence of 23.6% among cases of non-traumatic paraparesis or tetraparesis (Wu et al., 2013).

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In peripheral nervous system disorders, Guillain-Barré syndrome is a relatively more frequent cause with an incidence of 1–2 per 100,000 person-years (average 1.2) and a prevalence of about 1.1 per 100,000 population (Sejvar et al., 2011). Vasculitic neuropathy has an incidence of 4.2 per 100,000 for lumbosacral radiculoplexus neuropathy and accounts for 28% of all vasculitic neuropathy in patients with ANCA-associated vasculitis (Collins et al., 2011).

In neuromuscular joint disorders, myasthenia gravis had an incidence of 5.3 per 100,000 person-years with a prevalence of 17.3 per 100,000 (or 173.3 per million) in the United States in 2021 (Carr et al., 2010; Murai et al., 2024). Lambert-Eaton myasthenic syndrome is a much rarer condition with a prevalence of only 0.3 per 100,000 population (Titulaer et al., 2017).

Demyelinating diseases such as multiple sclerosis have a global prevalence of 36 per 100,000 population with significant regional variations: 133 per 100,000 in Europe and 112 per 100,000 in the Americas (GBD 2019 Multiple Sclerosis Collaborators, 2023). Neuromyelitis optica, a rarer condition, has an incidence of 0.053–0.4 per 100,000 per year and a prevalence of 0.5–5 per 100,000 population (Mealy et al., 2012).

In muscle disorders, idiopathic inflammatory myopathies (IIMs), including polymyositis and dermatomyositis, have an incidence of 0.14–1.0 per 100,000 per year, with dermatomyositis recorded at 1.4 per 100,000 and polymyositis at 3.8 per 100,000 in the United States based on data from 2003–2008 (Bernatsky et al., 2009). Gender distribution shows a predilection in females with a female-to-male ratio of about 2:1 (Bernatsky et al., 2009; Lundberg et al., 2012). IIM is a heterogeneous group of autoimmune diseases characterized by progressive symmetrical proximal muscle weakness, increased muscle enzymes (especially creatine kinase), myopathic changes in electromyography, and inflammatory infiltrates in muscle biopsies (Lundberg et al., 2012). Dermatomyositis has distinctive skin manifestations such as heliotrope rash, Gottron's papules, and the V-sign or shawl sign that distinguishes it from polymyositis (Furst et al., 2012).

In the context of cases of acute or subacute tetraparesis in young women, IIM needs to be considered as one of the main differential diagnoses, given the obvious gender predilection, onset at a productive age, as well as clinical manifestations that can resemble various other neuromuscular disorders (Bernatsky et al., 2009; Lundberg et al., 2012). Early identification and appropriate immunosuppressive treatment in IIM are essential to prevent long-term complications such as permanent weakness, dysphagia with aspiration risk, interstitial lung involvement, as well as related malignancies, especially in dermatomyositis (Furst et al., 2012). Understanding the spectrum of clinical and epidemiological manifestations of IIM is an important basis for the diagnosis and management approach of patients with suspected inflammatory myopathy.

Idiopathic Inflammatory Myopathies (IIM) or also known as myositis, is a group of heterogeneous autoimmune diseases that affect the structure, metabolism, and function of skeletal muscle. The disease is characterized by chronic muscle inflammation with varied clinical manifestations, therapeutic response, and prognosis (Lundberg, 2021). The main clinical symptom is muscle weakness; however, patients may also complain of other symptoms such as muscle pain and stiffness, cramps, as well as extramuscular symptoms (Nagy & Veerapaneni, 2025; van der Kooi & de Visser, 2014). Myopathy can be caused by impaired muscle tissue integrity or impaired muscle metabolic stability triggered by genetic factors, medications, toxins, bacterial/viral infections, inflammation, electrolyte balance disorders, and hormonal imbalances (Khoo et al., 2023; Nagy & Veerapaneni, 2025).

The classification of Idiopathic Inflammatory Myopathies (IIM) consists of dermatomyositis (DM), polymyositis (PM), necrotic myopathy, and Inclusion Body Myositis (IBM). Diagnosis enforcement is carried out based on anamnesis, physical examination, special laboratory examinations such as muscle enzyme levels, electromyography examinations, MRIs, and muscle biopsies (Ashton et al., 2021; Connolly et al., 2024; Nandrekar & Patil, 2025). Autoantibodies specific to myositis have an important role in the classification of IIM subtypes. Primary therapy consists of glucocorticoids, immunosuppressants, intravenous

immunoglobulins, and biological therapies such as Rituximab. Recent research focuses on the latest biomarkers, target therapies, and the role of immune system modulation. The prognosis of myositis varies, so that in future studies focused on more effective and personalized therapeutic approaches to myositis are needed (Connolly et al., 2024; Nandrekar & Patil, 2025).

Case

A female patient, Mrs. SF, 27 years old, domiciled in Gresik, East Java, came to the Rheumatology Polyclinic of Dr. Soetomo Hospital with the main complaint of weakness in both shoulders and thighs for 5 months. This weakness is felt to be even more burdensome. Initially, the patient was suspected of suffering from Guillain-Barré Syndrome (GBS), but after an examination at the Neurology Poly, the patient's condition was not consistent with the disease, so the patient was referred to the Rheumatology Poly. Patients also complain of pain in the muscles of the upper arm and thighs that are missing. The patient said the back of the neck also felt stiff. Patients feel hampered in carrying out daily activities such as raising hands, standing, and walking.

Patients did not complain of fever, seizures, paralysis, swallowing disorders, skin rashes, or pain and swelling in the joints. The patient complained of coughing and shortness of breath since >3 months. The patient was suspected of being infected with pulmonary TB, but after a sputum examination was carried out, negative results were obtained. The patient has never experienced similar complaints before. None of the patient's family members have experienced similar complaints.

On physical examination, conscious consciousness was obtained with blood pressure of 106/74 mmHg, pulse 106 times/minute, breathing 22 times/minute, temperature 37 °C, and SO2 99% with a nasal cannula of 3 lpm. Examination of the head and neck did not find icteric sclera, anemical conjunctiva, cyanotic lip, nasal lobe breathing, or neck muscle retraction. The pressure of the jugular vein does not increase, and there is no enlargement of the lymph nodes of the neck. On examination of the thoracic region, symmetrical chest wall movements were obtained, no intercostal or supraclavicular retraction, bilateral hemithoracic sonor percussion, bilateral hemithoracic vesicular breathing sound, no wheezing, but rhonchi were found under both lung chambers. On the cardiac examination, it was found that the cord was located between the ribs to the 5th $\frac{1}{3}$midclavicula line sinistra, the heart sound S1 S2 was single, there was no murmur, and there was no gallop. Examination of the abdominal region showed convex, normal bowel noise, no pectoral pain, and no palpable organomegaly. Examination of the upper and lower extremities found that the areas were warm, dry, red, and there was no edema.

In the examination of the field of Rheumatology and Neurology, motor strength on the four proximal sides was found to be 2, which showed that the extremities could not withstand gravity and were sesoric within normal limits. Meanwhile, on the distal side, motor strength is still obtained at 5, where it can still withstand a pull.

Laboratory examination on April 10, 2025 at Dr. Soetomo Hospital obtained results of Hb 11.8 g/dL, Hematocrit 35.3%, MCV 77.8 fl, MCH 26 pg, Leukocytes 17060/mm3 (Neut 73.4%, Lymph 18.6%), Platelets 501000/mm3, LED 92, BUN 5 mg/dL, Serum Creatinine 0.15 mg/dL, SGOT 141 U/L, SGPT 87 U/L, Albumin 4.52 g/dL, GDA 106 mg/dL, Potassium 4.8 mmol/L, Sodium 141 mmol/L, Chloride 101 mmol/L, CRP 0.79, Procalcitonin 0.05, ANA 11.1, TSH 0.434, HbSAg, anti-HCV, and HIV non-reactive; Blood Gas Analysis: pH 7.4, pCO2 46 mmHg, pO2 86 mmHg, Be 3.7 mmol/L, SO2 97% (O2 free air) and HCO3- 28.5 mmol/L; Urinalysis: clear yellow color, BJ 1.024, pH 6.0, glucose, ketones, nitrites, erythrocytes, leukocytes, bilirubin, and urobilinogen negative, bacteria 1.69, albumin/ protein 80/ +1. The patient was also examined for the enzyme creatinine kinase on December 6, 2024, with results of 11931 (26 – 192).

An electromyography examination (EMG) on November 25, 2024, showed the presence of secondary myopathic lesions with signs of neuropathy in the form of denervation in the bilateral m. deltoid and left rectus femoris m. The patient underwent two thoracic X-rays on March 11, 2025, and March 24, 2025, with the results of an image of pneumonia infiltrates in the perihilar and pericardial

areas, as well as bilateral pleural effusion.

The initial assessment of patients based on anamnesis, physical examination, and supporting examinations was suspect of Idiopathic Inflammatory Myopathies (IIM) and Bilateral Pneumonia and suspected Interstitial Lung Disease (ILD). The patient undergoes hospitalization for initial diagnosis and management. Patients are planned to perform myopathic profile serological examinations, x-rays and MRI of the pelvis and femur, muscle biopsy, sputum culture, and thoracic HRCT. The patient's initial management plan was a high-calorie and protein diet of 1900 kcal/day, infusion of methylprednisolone 500 mg/day for 3 days, and symptomatic therapy in the form of codeine and oral acetylcysteine.

On the second day of treatment, April 11, 2025, the patient still complained of weakness in both shoulders and thighs, but it was not severe. Vital signs and physical examination within normal limits. The patient underwent a thoracic X-ray evaluation with a picture of bilateral pneumonia that seemed burdensome. On the examination of the femur and pelvis, bilateral coxae valga was obtained. MRI examination of the femur and pelvis showed the presence of abnormal hyperintensity in the anterior, medial, and posterior compartments, with the conclusion of suspect Idiopathic Inflammatory Myopathies. Microbiological examination of sputum culture found the presence of PMN cells 2+, epithelial cells 2+, gram-negative bacilli 2+, gram-positive cocci 4+, yeast, with the growth of germs found Streptococcus viridans and Candida albicans that are sensitive to Fluconazole, Voriconazole, Caspofungin, Micafungin, Amphotericin B, and Flucytosine. The patient received pharmacological management in the form of an infusion of Methylprednisolone 500 mg/day (first day), Codeine 10 mg/8 hours, and Acetylcysteine 200 mg/8 hours.

On the fifth day of treatment, April 14, 2025, the patient felt weakness in both shoulders and thighs had reduced after 3 days of Methylprednisolone 500 mg therapy. No cough and tightness. Vital signs and physical examination within normal limits. The results of the myopathic profile serology examination showed the results of myositis-specific antigens, namely NXP 1+ and SRP 3+. Thoracic HRCT examination found a picture of pneumonia and excluded interstitial lung disease. Patients received follow-up therapy in the form of Cyclophosphamide infusion 500 mg/day (single dose), Methylprednisolone injection 40 mg/day, Codeine 10 mg/8 hours, and Acetylcysteine 200 mg/8 hours. Collaboration with the Department of Medical Rehabilitation is carried out as one of the non-pharmacological management efforts. Physical exercises were carried out in the form of posture correction, self-mobilization, breathing control, and chest expansion related to pneumonia, as well as exercises to improve the Range Of Motion (ROM).

Eighth day treatment, April 17, 2025, muscle weakness improved. The patient is planned to be an outpatient after a muscle biopsy. Vital signs and physical examination within normal limits. Muscle strength has increased to 4/4/4/4. The evaluation of liver function and creatinine kinase enzymes improved, namely SGOT 141 → 66, SGPT 87 → 74, and creatinine kinase 11931 → 852. The biopsy of m. vastus medialis femur dextra provides an overview of lymphocyte infiltration in the endomysium and perivascular. The picture supports the diagnosis of Idiopathic Inflammatory Polymyositis and rules out suspected necrotic autoimmune myopathy, juvenile myositis, inclusion body myositis, and dermatomyositis. Patients received antibiotic therapy infusion of Levofloxacin 500 mg/ day (since April 15, 2025), Methylprednisolone injection 40 mg/day (April 14 – April 18, 2025), Codeine 10 mg/ 8 hours, and Acetylcysteine 200 mg/ 8 hours. KRS patients on the ninth day and planned to be controlled in the Rheumatology Poly.

The patient is planned for control in the Rheumatology Poly on April 21, 2025. The evaluation of the patient's condition during control was that the patient felt that the complaints were getting better, the weakness was further overcome with PO MP 16mg-8mg-8mg therapy, PO Fluconazole 1x 400 mg, which was lowered to 1x 200 mg, and PO Levofloxacin 1x 500 mg. Patients are then planned to cycle 500mg once every 1 month for 6 cycles starting May 14, 2025.

Discussion

Tetraparesis is a motor disorder that involves

weakness of all four extremities and can signal the presence of a serious illness that threatens the patient's safety. A quick identification process is especially important because this condition often requires critical intervention. The management of tetraparesis is greatly influenced by its underlying causes; Therefore, early identification and determination of etiology are the first steps that determine the prognosis and success of therapy (Oliveira R, 2021).

Some of the serious underlying diseases of tetraparesis include disorders of the brain (such as bilateral stroke of ACA with impaired consciousness and motor skills), brainstem abnormalities (such as locked-in syndrome due to basilar occlusion or central pontine myelolysis), to disorders in the spinal cord (e.g., spinal infarction or transverse myelitis). On the other hand, tetraparesis can also appear as a result of peripheral nervous system disorders, such as Guillain-Barré syndrome, vasculitic neuropathy, disorders in neuromuscular joints (myasthenia gravis, Eaton-Lambert, or toxic), and muscular disorders (such as inflammatory, endocrine, metabolic, as well as toxic effects) (Oliveira R, 2021).

The accuracy of distinguishing the location and type of disturbance (CNS vs PNS) greatly affects the next management. Each etiology has its own treatment protocol, so systematic differential diagnosis is an important foundation in the treatment of tetraparesis patients. This condition often marks severe disease and allows for a risk of decreased vital function, so early recognition and action are highly recommended for better clinical outcomes (Oliveira R, 2021).

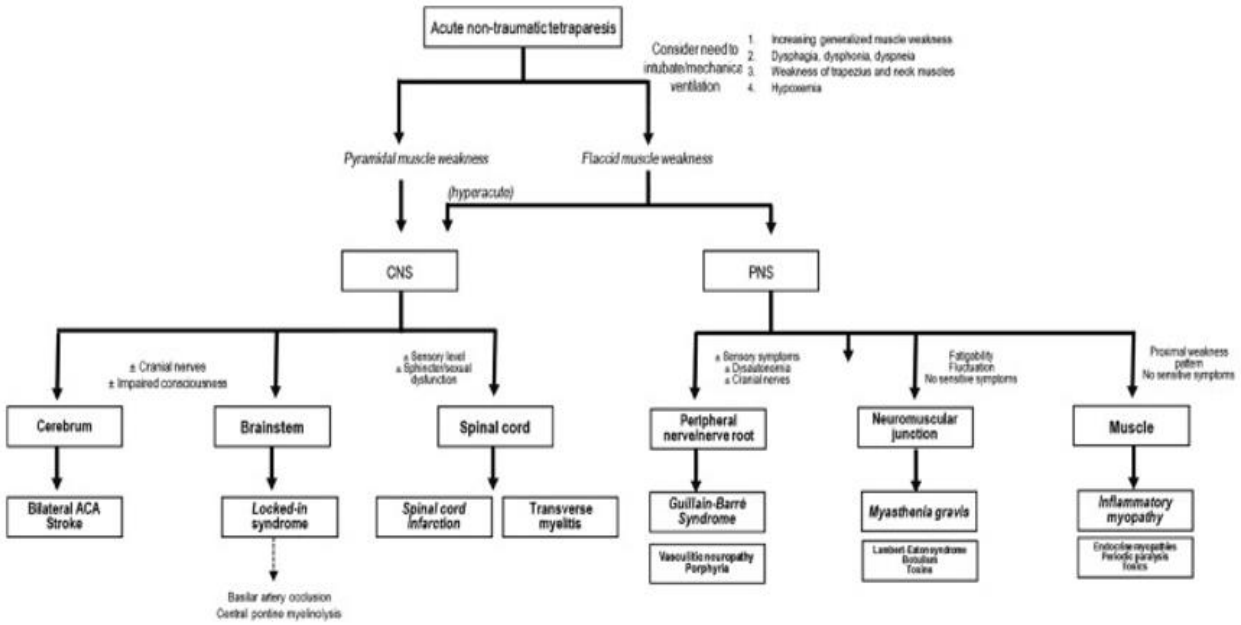


Figure 1. An alternative diagnosis approach in cases of non-traumatic acute tetraparesis (Oliveira R, 2021)

Idiopathic Inflammatory Myopathies (IIMs) are a group of heterogeneous autoimmune diseases with chronic inflammatory characteristics of the muscles and have varied clinical manifestations, therapeutic responses, and prognosis (Lundberg et al., 2021). This group of diseases is classified into dermatomyositis (DM), polymyositis (PM), necrotic myopathy, inclusion body myositis (IBM), and several new subtypes such as Immune-Mediated Necrotizing Myopathy (IMNM), anti-synthetase syndrome, juvenile DM, amyopathic DM, and malignant associated myositis (Tanboon & Nishino, 2019; Firestein et al., 2021). A typical clinical presentation is usually symmetrical proximal muscle weakness. Patients may also complain of extramuscular symptoms such as respiratory, cardiovascular, musculoskeletal, and gastrointestinal disorders (Adhoubi et al., 2024).

The prevalence, incidence, and characteristics of IIM

vary from country to country. Studies in Singapore and Israel reported IIM incidences of 7.7 and 2.18 cases per 1 million population per year (Koh et al., 1993; Benbassat et al., 1980). A systematic review by Meyer et al. reported that the incidence of IIM was 1.16 – 19 cases per 1 million population per year, with a prevalence of 2.4 – 33.8 cases per 100000 (Meyer et al., 2014). The prevalence of IIM in Thailand is similar to the prevalence of IIM worldwide, which is 13.93 per 100000 (Onchan et al., 2024).

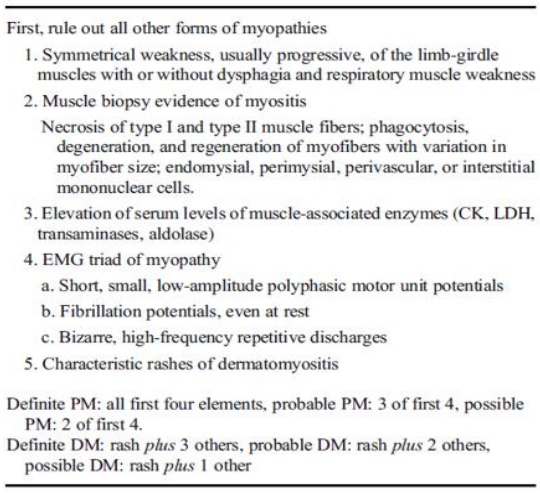
Women dominate IIM cases in Thailand with a ratio of 1.6:1 to men (Onchan et al., 2024). This is in line with a study by Lynn et al., which showed similar findings but with a higher ratio, which is 4:1 (Lynn et al., 2005). Findings from several studies collected show that PM and DM are more suffered among women, while IBM is more prevalent among men (Onchan et al., 2024). IIM generally occurs in the fourth to sixth decade. Research by Onchan et al., in Thailand, reported the highest prevalence in the age range of 60 – 69 years (Onchan et al., 2024). The prevalence of IBM is higher in older age groups compared to PM and DM. The IBM subtype is more prevalent in men aged >50 years, while DM is more prevalent in the <50-year-old age group than in PM (Gazeley & Cronin, 2011; Jam et al., 2018).

The heterogeneity of myositis makes it difficult to investigate predisposing factors. Epidemiological studies show that DM is more prevalent in the East Asian population. Necrotic myositis is 150 times more common in the American Indian population receiving statin therapy (Musai et al., 2024). Environmental factors, such as areas that are exposed to high UV or are close to the equator, have a higher frequency of DM than other myositis subtypes. Exposure to dust and environmental pollutants also increases the risk of myositis (Love et al., 2009). Gastrointestinal and lower respiratory tract infections have been reported to be associated with the pathogenesis of the occurrence of PM and DM. Other viral infections, such as HIV, HTLV, and HCV, are also thought to be related to other subtypes of myositis, although their role in their pathogenesis is unclear (Musai et al., 2024).

The development of myositis is influenced by complex interactions of microbial infections, drugs, genetic susceptibility, and environmental exposure. These factors contribute to the formation of myositis-specific autoantibodies in muscle tissue. Recent research links the pathogenesis of myositis to the intracellular action of autoantibodies that interfere with its specific autoantigen function. This is what causes inflammation and toxicity of muscle cells (Musai et al., 2024).

A 27-year-old woman came in with complaints of weakness in the bilateral proximal muscles (shoulder and thigh muscles) for 5 months. The patient lives in Gresik, East Java, which has high exposure to UV rays, even though the patient works indoors every day. No nuclear family has such complaints as patients. The patient has a history of lower airway infection, suspected of TB infection.

The classification criteria of Bohan and Peter (Figure 1), which were first published in 1975, are used as the classification and diagnosis criteria of IIM to date. DM and PM are distinguished by the presence of a typical skin rash in DM. There are definitions of diagnosis for "definite", "probable", and "possible" as well as exclusion criteria to eliminate other diseases similar to IIM. These classification criteria have been criticized for including nonspecific myositis features (myopathic electromyography), obscure exclusion criteria, and not specifying the exact number of features needed to meet certain specific criteria (Leclair & Lundberg, 2018).



First, rule out all other forms of myopathies

1. Symmetrical weakness, usually progressive, of the limb-girdle muscles with or without dysphagia and respiratory muscle weakness
2. Muscle biopsy evidence of myositis
   Necrosis of type I and type II muscle fibers; phagocytosis, degeneration, and regeneration of myofibers with variation in myofiber size; endomysial, perimysial, perivascular, or interstitial mononuclear cells.
3. Elevation of serum levels of muscle-associated enzymes (CK, LDH, transaminases, aldolase)
4. EMG triad of myopathy
   a. Short, small, low-amplitude polyphasic motor unit potentials
   b. Fibrillation potentials, even at rest
   c. Bizarre, high-frequency repetitive discharges
5. Characteristic rashes of dermatomyositis

Definite PM: all first four elements, probable PM: 3 of first 4, possible PM: 2 of first 4.
Definite DM: rash *plus* 3 others, probable DM: rash *plus* 2 others, possible DM: rash *plus* 1 other

Figure 2. Bohan and Peter's criteria for DM and PM (Leclair & Lundberg, 2018)

The 1991 revised Dalakas diagnosis criteria (Figure 2) have a more complete description than Bohan and Peter's criteria, including histopathological features found in DM, PM, and IBM (Dalakas & Hohlfeld, 2003). The classification of IIMs has been a matter of debate for decades. Previously unvalidated classification criteria, some studies involved patient data, a small number of cohort patients, single-centre, and did not involve a control group. The classification criteria published by EULAR/ACR (Figure 3) aim to distinguish IIM from similar diseases and categorize IIM into subtypes with available clinical and laboratory features. These classification criteria have good sensitivity and specificity, especially when the patient undergoes a muscle biopsy (Lundberg et al., 2017; Khan et al., 2019).

Criterion	Polymyositis		Myopathic dermatomyositis		Amyopathic dermatomyositis
	Definite	Probable	Definite	Probable	Definite
Myopathic muscle weakness	Yes*	Yes*	Yes*	Yes*	No†
Electromyographic findings	Myopathic	Myopathic	Myopathic	Myopathic	Myopathic or non-specific
Muscle enzymes	High (up to 50 times normal)	High (up to 50 times normal)	High (up to 50 times normal) or normal	High	High (up to 10 times normal) or normal
Muscle-biopsy findings	Primary inflammation, with the CD8/MHC-1 complex and no vacuoles	Ubiquitous MHC-I expression, but no CD8-positive infiltrates or vacuoles‡	Perifascicular, perimysial or perivascular infiltrates; perifascicular atrophy	Perifascicular, perimysial or perivascular infiltrates; perifascicular atrophy	Non-specific or diagnostic for dermatomyositis (subclinical myopathy)
Rash or calcinosis	Absent	Absent	Present	Not detected	Present

*Myopathic muscle weakness, affecting proximal muscles more than distal ones and sparing eye and facial muscles, is characterised by a subacute onset (weeks to months) and rapid progression in patients who have no family history of neuromuscular disease, no exposure to myotoxic drugs or toxins, and no signs of biochemical muscle disease. The myopathic weakness has a pattern distinct from that seen in inclusion-body myositis (table 1). †Although strength is apparently normal, many patients have new onset of easy fatigue, myalgia, and reduced endurance. Careful muscle testing may reveal mild muscle weakness. ‡If such a patient has the clinical phenotype of sporadic inclusion-body myositis, the diagnosis will be probable inclusion-body myositis; a repeat biopsy is indicated.

Figure 3. IIM diagnosis criteria (Dalakas & Hohlfeld, 2003)

When no better explanation for the symptoms and signs exists these classification criteria can be used

Variable	Score	
	No muscle biopsy	With muscle biopsy
Age of onset of first symptom assumed to be related to the disease ≥ 18 and < 40 years	1.3	1.5
Age of onset of first symptom assumed to be related to the disease ≥ 40 years	2.1	2.2
Muscle weakness		
Objective symmetric weakness, usually progressive, of the proximal upper extremities	0.7	0.7
Objective symmetric weakness, usually progressive, of the proximal lower extremities	0.8	0.5
Neck flexors are relatively weaker than neck extensors	1.9	1.6
In the legs, proximal muscles are relatively weaker than distal muscles	0.9	1.2
Skin manifestations		
Heliotrope rash	3.1	3.2
Gottron's papules	2.1	2.7
Gottron's sign	3.3	3.7
Other clinical manifestations		
Dysphagia or esophageal dysmotility	0.7	0.6
Laboratory measurements		
Anti-Jo1 autoantibody present	3.9	3.8
Elevated serum levels of CK or LDH* or ASAT/AST/SGOT* or ALAT/ALT/SGPT*	1.3	1.4
Muscle biopsy features—presence of:		
Endomysial infiltration of mononuclear cells surrounding, but not invading, myofibres		1.7
Perimysial and/or perivascular infiltration of mononuclear cells		1.2
Perifascicular atrophy		1.9
Rimmed vacuoles		3.1

Figure 4. EULAR/ ACR classification criteria for adult and juvenile IIMs (Lundberg et al., 2017)

The clinical presentation of all IIM subtypes except IBM is symmetrical proximal muscle weakness. IBM patients show different symptoms, namely muscle atrophy and weakness of the distal and peripheral muscles (Ashton et al., 2021). Patients may find it difficult to stand up from a chair, climb stairs, or dry clothes. IIM patients may exhibit acute, subacute, or slow symptoms and a variety of nonspecific symptoms such as fatigue, malaise, weight loss, myalgia, or atralgia that can resemble other disorders and result in a delay in diagnosis (Yazici et al., 2002).

Respiratory disorders and weakness of the pharyngeal muscles that cause dysphonia, dysphagia, shortness of breath, and susceptibility to pneumonia infections are often found in severe IIM conditions (Malik et al., 2016). Typical skin rashes are heliotrope rash (a red/purple periorbital erythematous rash on one or both eyelids, often accompanied by edema) and Gottron's pappules (reddish, reddish or bright red papules scattered on the dorsal metacarpal or interphalang; pathognomonic for the diagnosis of DM; called Gottron's rash if there is only a macular rash without papules) is often encountered in DM cases. Other symptoms can be calsinosis, but they usually appear in pediatric IIM patients. (Oldroyd et al., 2017; Firestein et al., 2021; Nandrekar & Patil, 2025).

Table 1. Symptoms and clinical picture in IIM subtypes (Firestein et al., 2021)

	DM	**PM**	**IBM**	**NM**
Age	Children/adults	Adult	> 50 years	Adult
Onset	Subakut	Subakut	Kronik	Subakut
Muscle weakness	Proximal	Proximal	Selective patterns	Proximal
Simetris	Ya	Ya	No	Ya
Systemic symptoms	Ya	Ya	Ya	Ya
Skin lesions	Ya	No	No	No
Kalsinosis	Ya	Infrequently	No	No
Related systemic autoimmune	Ya	Ya	Ya	No
Related to violence	Ya	Unknown	Unknown	Unknown
Enzyme serum	Normal-tinggi	Normal-tinggi	Normal-tinggi	Tall
EMG abnormal	Ya	Ya	Ya	Ya
Biopsi otot abnormal	Atrofi perifasikular, deplesi kapiler, patchy class I MHS expression, and microinfarcts	Invasi sel limfosit CD8+ di non-necrotic fibers, class I MHC expression on fibers	Invasi sel limfosit CD8+, ekspresi MHC, vacuolated fibers, dan tubulofilamentous inclusions in fibers	Nekrotik dan regenaris serat otot, upregulation of class I in occasional fibers

Note: PM rarely appears in children

Selective pattern of IBM muscle weakness: weakness begins in the finger flexors, wrist flexors/extensors, and quadriceps femoris.

Systemic symptoms: DM and PM can be accompanied by dysphagia, synovitis, and ILD; IBM and NM are accompanied by dysphagia.

Typical skin lesions: Gottron's papules/rash,

heliotropic rash; only on DMs.

Calcinosis is only in children.

DM and PM can overlap with scleroderma, Sjogren's syndrome, LES, RA, and MCTD.

IBM may be associated with Sjogren's syndrome, but it is rarely associated with other autoimmune diseases.

Serum Enzymes: CK, SGOT, SGPT, LDH, and aldolase.

The levels of the enzyme creatinine kinase vary

depending on the IIM subtype. Levels of this enzyme can increase up to 50 times in 60 – 70% of DM cases. An increase of 5 – 50 times was obtained in PM. Levels of this enzyme can be used as biomarkers of therapeutic response and relapse conditions. Other muscle enzymes, such as LDH, SGOT, SGPT, and aldolase, are less sensitive and their levels may be normal (Malik et al., 2016; Ashton et al., 2021). The presence of connective tissue disease antibodies can be suspected that the myopathy that occurs is secondary to connective tissue disease. Antinuclear antibodies (ANA) were detected in 24% – 60% of DM cases, 16% – 40% of PM cases, and 20% in IBM (Malik et al., 2016).

Patients have with major complaints of weakness in both shoulders and thighs for 5 months. The weakness of both shoulders and thighs is felt to be burdensome, so that it is difficult for patients to carry out daily activities such as raising their hands, standing, and walking. Patients also complain of lost muscle pain. The patient's neck feels stiff. Patients did not complain of fever, seizures, paralysis, swallowing disorders, skin rashes, or pain and swelling in the joints.

A general physical examination showed normal vital signs, but on thoracic physical examination, a rhonki sound was found in the $\frac{1}{3}$ Lower region of the second lung field. Laboratory tests showed an increase in leukocytes, LEDs, and CRP – suspected infectious and inflammatory processes. Muscle enzymes also increased, namely SGOT 141, SGPT 87, and creatinine kinase 11931. ANA and TSH levels are within normal limits, so that the possibility of underlying autoimmune diseases can be ruled out. The patient's history of chronic cough was initially suspected to be a TB infection; however, this possibility could be excluded after the sputum gene Xpert test was negative. Based on the findings of the anamnesis, physical examination, and preliminary laboratory examination, the patient was diagnosed with suspected IIM and bilateral pneumonia, and interstitial lung disease. Patients are planned to perform myopathic profile serological examinations, pelvic and femur MRI, muscle biopsies, and thoracic HRCT.

Myositis-specific antibodies play an important role in the characterization of IIM subtypes and have diagnostic and prognostic value. These antibodies are detected in 85% of adult IIM patients (Mariampillai et al., 2018). Myositis-specific antibodies consist of cytoplasmic antibodies against Mi-2 and Mas antigens, antibodies targeting translational proteins such as tRNA synthetase, anti-SRP, transcriptional intermediary factor-1 gamma (TIF-1; anti-155/140 Ab), and melanoma differentiation-associated gene-5 (MDA5; anti-CADM140 Ab) (Malik et al., 2016).

Nerve conduction studies (NCV) and electromyography (EMG) can be performed to determine the presence of myopathy processes and exclude other neuromuscular or neurological conditions. A common description of EMG is an "irritable myopathic process" with the following characteristics: (1) increased spontaneous and insertive activity with the potential for fibrillation – sharp positive waves, small myopathic units, and the absence of large neurogenic units. Muscle fibrosis results in decreased insertive activity 2), short duration, and low amplitude in polyphasic Motor Unit Action Potentials (MUAPs) (Amato & Barohn, 2009).

Magnetic Resonance Imaging (MRI) can demonstrate abnormal signals in the muscles due to inflammation, edema, or replacement of muscles with fibrotic tissue. MRI can also help clinicians to determine the location of a muscle biopsy (Amato & Barohn, 2009). T2 fat suppression and short-tau inversion recovery (STIR) describe muscle edema as a high uptake signal that indicates inflammation. Fat infiltration in T1 also includes high uptake signals as a sign of chronic impairment (Asthon et al, 2021).

Muscle biopsy is an important modality in the diagnosis of IIM. Muscle biopsy can show inflammation, identify clinics based on IIM subtypes, and exclude other possible myopathies (Figure 2 and Table 1) (Lundberg et al., 2021). Other examinations to look for complications of the cardiovascular system (electrocardiography, echocardiography, and pulmonary functional examination) need to be performed on all IIM patients. High-Resolution Computed Tomography (HRCT) of the thorax should be performed when there is a suspicion of interstitial pulmonary disease. The malignancy can be related to DM (25%), PM (10%), and also to necrotic myopathy. Generally, patients are diagnosed in the first to fifth year. There is no consensus or guideline regarding

malignancy in IIM until now; however, patients still need periodic malignancy screening (Malik et al., 2016; Ashton et al., 2021).

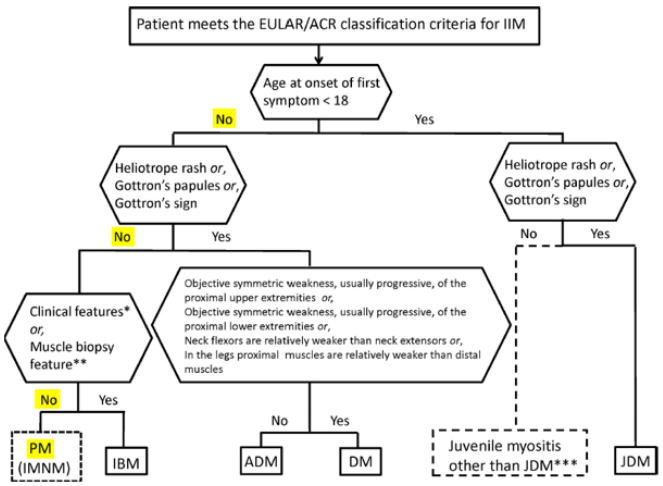


Figure 5. Classification of IIM subtypes. patients must meet the EULAR/ACR classification criteria (IIM probability ≥55%) (Lundberg et al., 2017)
Note: IMNM patients include patients with the PM subtype

For the IBM classification, meeting one of the following: *flexor finger weakness and poor therapeutic response, or **muscle biopsy found rimmed vacuole.

Juvenile myositis, in addition to juvenile dermatomyositis, is determined by expert opinion.

Serological examination of the patient's myopathic profile obtained the results of NXP2 +1 and SRP 3+; both antigens are myositis-specific antigens. NXP2 antigens are associated with dermatomyositis, myositis-induced cancers, and juvenile dermatomyositis, while SRP 3 antigens are associated with immune-related polymyositis and necrotic myopathy. The patient also underwent an EMG examination with the conclusion of secondary myopathic lesions – a sign of neuropathy in the form of denervation in the bilateral muscles. Deltoid and the left m. rectus femoris. MRI examination of the femur and pelvis showed the presence of abnormal hyperintensity in the anterior, medial, and posterior compartments, with the conclusion of suspect Idiopathic Inflammatory Myopathies. Biopsy examination was performed on the vastus medialis muscle of the femur dextra according to the abnormalities found on the MRI. The results of the muscle biopsy were in the form of lymphocyte infiltration in the endomysium and perivascular. The picture supports the diagnosis of Idiopathic Inflammatory Polymyositis and rules out suspected necrotic autoimmune myopathy, juvenile myositis, inclusion body myositis, and dermatomyositis. The patient also underwent a thoracic HRCT examination to rule out the possibility of interstitial lung disease, and the results showed a picture of bilateral pneumonia without a picture of interstitial lung disease. The patient was diagnosed with Idiopathic Inflammatory Myositis subtype polymyositis according to the EULAR/ ACR classification criteria – the probability of the patient's IIM was 85% (Figure 3 and Figure 4).

The purpose of IIM management is to restore muscle strength, overcome inflammation, and prevent organ damage. The management of IIM requires multidisciplinary cooperation from the fields of rheumatology, neurology, pulmonology, dermatology, and medical rehabilitation (occupational and speech therapy). Generally, necrotic myositis is more resistant to immunosuppressants than DM and PM, especially when it is triggered by statins and there is an underlying malignancy. The majority of IBM patients have a poor response to immunotherapy (Malik et al., 2016).

High-dose corticosteroids are the main line of therapy for DM, PM, and necrotic myositis. The initial dose of Prednisone is 0.5 – 1 mg/kg/ day (60 – 100 mg/day), then the dose is lowered after 4 – 6 weeks, depending on the patient's clinical condition. If, after administration of high doses of Prednisone, the response is inadequate, then it is necessary to think about the possibility of a diagnosis of IBM or muscular dystrophy. The addition of second-line agents is based on the severity of the disease, initial response to Prednisone, relapse conditions, and the risk of steroid complications (osteoporosis, diabetes). Immunosuppressive drugs are generally initiated at the same time as the initiation of steroid therapy. The therapy of choice is Methotrexate (MTX), Azathioprine (AZA), or Mycophenolate Mofetil (MMF) (Malik et al., 2016; Oldroyd et al., 2017).

Aggressive therapy is necessary in refractory conditions and severe diseases, i.e., severe muscle weakness, dysphagia, or drastic weight loss. Intravenous methylprednisolone can be administered at a dose of 500 – 1000 mg/day for 3 consecutive days, followed by an oral prednisone tapering dose. Cyclophosphamide works by blocking the proliferation of T cells and B cells. Cyclophosphamide is used when the patient fails to respond to all therapies or there is severe organ damage. Cyclophosphamides 0.6 – 1 g/m2 were administered after intravenous hydration, antiemetic, and Mesna. Intravenous cyclophosphamide is given weekly for 6 – 12 months. Therapies for other refractory and severe conditions are intravenous immunoglobulin (IVIG), Rituximab, Tacrolimus, TNF-α inhibitors, and Cyclosporin A. Emerging therapies for IIMs include Anankira, Belimumab, Alemtuzumab, and Sifalimumab (Malik et al., 2016; Nandrekar & Patil, 2025; Firestein et al., 2021).

Non-pharmacological therapy in the form of physical therapy aims to avoid complications from inflammation and muscle damage. Chronic inflammation results in muscle atrophy due to a lack of physical activity and is exacerbated by the side effects of long-term glucocorticoid use. The combination of pharmacological and non-pharmacological therapies can restore and improve patient performance and prevent exacerbations. Physical therapy can be started 4 weeks after initiation of immunosuppressive agents (Firestein et al., 2021). IIM outcome measurement needs to be done because myositis is a heterogeneous and complex disease. The assessment of IIM activities was carried out by assessing the visual analog scale (VAS) objectively and subjectively. Manual muscle examination to monitor muscle strength and function, serum evaluation of muscle enzymes (CK, SGOT, SGPT, LDH, Aldolase), and assess extramuscular disease activity score with certain indices such as myositis disease activity assessment VAS (MYOACT), myositis intention to treat activity index (MITAX) or physician's overall assessment of extramuscular disease activity on a visual analog scale are used to assess disease activity other than systemic symptoms or symptoms related to various organs such as skin rashes, joints, respiratory tract, muscles (Connolly et al., 2024; Firestein et al., 2021; Meyer et al., 2019).

The patient received initial therapy in the form of intravenous methylprednisolone 500 mg/day for 3 days with consideration of severe clinical symptoms (progressive bilateral proximal muscle weakness and impaired activity). The intravenous dose of methylprednisolone was then lowered to 40 mg/day on the fifth day of hospitalization, accompanied by the administration of 500 mg/day of Cyclophosphamide at a single dose. The patient also received intravenous Levofloxacin therapy 500 mg/24 hours to treat pneumonia infection. Evaluation of complaints of muscle weakness and pain improved from the fifth day of hospitalization. The evaluation of liver function and creatinine kinase enzymes improved, namely SGOT 141 → 66, SGPT 87 → 74, and creatinine kinase 11931 → 852. Patients also received non-pharmacological management in the form of physical exercises to correct posture, independent sitting mobilization, breathing control, and chest expansion exercises related to pneumonia, as well as exercises to improve and maintain limb ROM. The patient was discharged on the ninth day of hospitalization with Methylprednisolone 16 mg-16 mg-0, Levofloxacin 500 mg/ 24 hours, Acetylcysteine 200 mg/ 8 hours, Codeine 10 mg/ 8 hours. The patient is planned for control in the Rheumatology Poly on April 21, 2025. The evaluation of the patient's condition during control was that the patient felt that the complaints were getting better, the weakness was further resolved with PO MP 16mg-8mg-8mg therapy, PO Fluconazole 1x 400 mg > 1x 200 mg, and PO Levofloxacin 1x 500 mg. Patients are then planned to cycle 500mg once every 1 month for 6 cycles starting May 14, 2025.

Conclusion

A 27-year-old female patient with a major complaint of bilateral proximal muscle weakness in the upper and lower extremities was diagnosed with Idiopathic Inflammatory Myopathies Subtype Polymyositis. The enforcement of the diagnosis and classification of disease subtypes is based on anamnesis, physical examination, laboratory, radiology, and pathology in accordance with EULAR/ACR 2017. Patients received medicamentous therapy in the form of a pulse dose of intravenous methylprednisone 500 mg for 3 consecutive days and intravenous cyclophosphamid

500 mg single dose. Clinical complaints and evaluation of muscle enzymes (SGOT, SGPT, and creatinine kinase) improved on the fifth day of hospitalization. The patient was discharged with a good initial response to therapy and scheduled for routine check-ups at the Rheumatology Poly.

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