International Journal of Advances in Medicine
Katz A et al. Int J Adv Med. 2022 Jun;9(6):756-759
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20221361

Case Report

An emerging role of platelet-rich plasma and hyperbaric oxygen in the management of inclusion body myositis: a case report

Alan Katz[1], Kristin Thomson[1,2]*, Daniel Morgan[1]

[1]Hyperbaric Medical Solutions, NY, USA
[2]Stony Brook University, NY, USA

Received: 06 April 2022
Revised: 29 April 2022
Accepted: 05 May 2022

***Correspondence:**
Dr. Kristin Thomson,
E-mail: kthomson@hmshbot.com

ABSTRACT

Inclusion body myositis is an uncommon inflammatory myopathy that causes progressive muscle weakness. Patient management includes immunosuppressant therapy and nonpharmacologic therapies, like physical, occupational, and speech therapy. Standard treatment plans focus on the maintenance of muscle strength and function. Many patients do not respond to pharmacologic therapies and due to the progressive nature of this myopathy, patients eventually become debilitated. Hyperbaric oxygen therapy and platelet-rich plasma injections were provided as adjunctive therapy to a 70-year-old female patient with inclusion body myositis. After treatment, she had improvement in her muscle function and improved ambulation. This case study highlights the impact of adjunctive therapy in a patient with inclusion body myositis.

Keywords: Inclusion body myositis, Hyperbaric oxygen therapy, Hyperbaric oxygenation, Refractory wounds, Regenerative medicine, Platelet-rich plasma

INTRODUCTION

Inclusion body myositis (IBM) is an uncommon inflammatory myopathy that causes progressive muscle weakness. Sporadic IBM is categorized as an idiopathic inflammatory myopathy that occurs in older patient populations and is diagnosed more frequently in men.[1-3] The pathogenesis of IBM is not fully understood but has been associated with autoantibodies directed against cytosolic 5'-nucleotidase 1A(Cn-1A).[4]

The diagnosis of IBM can be challenging for both clinicians and patients. Diagnosis is made by a careful history and physical exam paired with laboratory findings and muscle biopsy. Muscle histopathology results typically yield rimmed vacuoles, and inflammatory infiltrates invading non-necrotic muscle. Autoantibodies against Cn-1A support the diagnosis.[1,4]

IBM can be misdiagnosed due to the unique insidious presentation of symptoms and overlapping presentation with other idiomatic inflammatory myopathies like polymyositis and dermatomyositis. Patients with IBM develop progressive muscle weakness and atrophy in the upper extremities and proximal lower extremity muscles. Characteristic muscle involvement includes the quadriceps muscles, finger flexors, and muscles involved with swallowing.[1] This weakness can lead to falls, dysphagia, and a decline in the ability to perform daily living activities.[2] Functional decline and mortality risk increase with age and progresses faster in patients after the age of sixty.[5]

Patient management includes a combination of immunosuppressant therapy and nonpharmacologic therapy, like physical, occupational, and speech therapy. Physical therapy and exercise are emphasized since it leads to demonstrated improvement in muscle strength.[2,6]

Treatment plans focus on the maintenance of muscle strength and function and maintaining daily living activities. IBM is refractory to some pharmacologic treatment plans and some patients use alternative forms of therapy to support their conventional treatment plans like acupuncture, massage, nutritional supplementation, and hyperbaric oxygen therapy.[1,7]

CASE REPORT

A 70-year-old female was diagnosed with IBM one year prior to consultation after experiencing progressive muscle weakness and difficulty walking. She was self-referred after being advised that the mainstay of her traditional therapy would be exercise and the use of supplements. Her earliest symptoms included the weakness of her hands and legs, and self-reported clumsiness and falling. Falls were occurring every three months due to muscle weakness. At presentation, she had a slightly elevated creatine kinase and her myositis-specific auto-antibodies were negative. Muscle biopsy, electromyogram, and MRI imaging confirmed the diagnosis. Her MRI reported disproportionate atrophy of the medial head of the gastrocnemius, long head of biceps femoris, and semimembranosus muscles bilaterally with muscle edema.

Figure 1: Four months post-treatment balance and strength photograph.

The influence of IBM on her daily activities was progressive. She reported that she cannot stand from a sitting position, climb stairs, lift her hands up to reach items on shelves, or have the strength to "pick up my baby grandkids." She had assistive devices like raised chairs, shower chairs, handrails, and a cane to help her with her daily activities. At the time of consultation, she was partially dependent on a wheelchair for mobility. She reported occasional mild choking symptoms.

Past medical history included hypertension, hypothyroidism, rheumatoid arthritis, and breast cancer with associated lumpectomy. Her rheumatoid arthritis was diagnosed ten years earlier than her IBM after having wrist and joint pain and the presence of cyclic citrullinated peptide antibody and rheumatoid factor on laboratory evaluation. She never received any immunosuppressant treatment and addressed her joint pain with holistic and dietary measures. The patient was a former smoker, uses alcohol sparingly, and reported no recreational drug use.

Her IBM therapeutic regimen included immunosuppressants like oral prednisone and the antimetabolite medication, methotrexate. She initiated physical, occupational, speech, acupuncture, and chiropractic therapies. The medications were eventually discontinued since they did not provide the anticipated therapeutic effects. Current medications at the time of referral were amlodipine, armour thyroid, folic acid, and nonprescription multi-vitamins.

A comprehensive three-month therapeutic plan was developed to be adjunctive to her conventional physical, occupational, and speech services. The plan incorporated platelet-rich plasma (PRP) injections and glutathione infusions paired with hyperbaric oxygen therapy (HBO). Glutathione is established as an antioxidant and neuromodulator, and 1g was infused twice weekly.[8] The hyperbaric therapy was performed at 2.4 ATA utilizing 100% oxygen with a five-minute air break every 20 minutes, five days a week for 90 minutes for a total of 60 sessions. Ultrasound-guided PRP was performed three times during the course of the HBO session, once after the first ten sessions of HBO, and again after twenty and sixty sessions. The edematous and atrophied rectus femoris and brachioradialis identified on MRI were the targets of the PRP therapy. More specifically, 5 ccs were divided and injected into each of the proximal, middle, and distal aspects of the rectus femoris muscles, and 2.5 ccs were divided and injected into each of the proximal, middle, and distal aspects of the brachioradialis muscles. The PRP was prepared utilizing a double spin technique. 18 cc of blood with 2cc anticoagulant was spun at 3400 rpm for 4 minutes, followed by a second spin of the isolated plasma and platelet solution at 3500 rpm for 2 minutes, yielding a leukocyte-poor, platelet-rich plasma with a concentration of 7x. Additionally, nutritional supplements were provided including mitochondrial support with an N-acetyl-L-Cysteine oral supplement as well as the supplement Mitochondrial NRG™.

The Inclusion Body Myositis Functional Rating Scale (IBMFRS)[9] was initially performed by her rheumatologist prior to initiating our therapy and was repeated by us at 16 weeks, 40 weeks, 60 weeks, and 16 weeks post-therapy. Scores were 24, 25, 32, 35, and 36 respectively (the maximum score is 40).

The IBMFRS is a validated tool that measures swallowing, handwriting, cutting food and handling utensils, fine motor skills, hygiene, turning in bed and adjusting covers, sitting to stand, walking, and climbing stairs. A higher score indicates higher functional ability.[9] In addition to her IBMFRS score, at the conclusion of her treatment plan, she reported improvement in her mobility and flexibility (Figure 1). She was able to easily walk thru a large airport,

get up from a chair without assistance, and hold her young grandchildren.

DISCUSSION

Sporadic inclusion body myositis is estimated to affect between 10 and 112 per million persons in the general population.[3] There is variation in the severity and progression of symptoms in patients. Multiple theorized mechanisms exist for IBM's pathogenesis. It is unknown whether IBM is a neurodegenerative or immune-mediated disease. The presence of cytotoxic CD8+ T cells and macrophages in non-necrotic muscle fibers surrounded by CD4+ T cells supports an autoimmune myopathy. Evidence of protein aggregates such as beta-amyloid, phosphorylated tau, and ubiquitin due to abnormal folding and disposal of proteins are associated with a neurodegenerative cause. Also, cN-1A, a protein involved in nucleic acid metabolism, is found in other autoimmune conditions such as Sjögren's syndrome, lupus erythematosus, and dermatomyositis.[2]

Treatment modalities for IBM have been aimed at targeting the inflammatory pathway but have been relatively ineffective.[6,7] Immunosuppressants, corticosteroids, and intravenous immunoglobulin have been shown to provide only temporary improvement in some patients.[3] Other novel pharmaceutical treatments for IBM such as rapamycin, bimagrumab, follistatin, and oxandrolone, were either not promising during trial or the research is still ongoing. IBM patients that receive physical therapy and engage in exercise regimens do preserve or increase muscle strength without causing harm.[2,3,7]

As adjunctive therapy, the use of hyperbaric oxygen therapy has safely decreased creatine kinase and improved symptoms associated with IBM. This includes the improved ability to swallow and the ability to flex fingers.[10] A case report demonstrated an IBM patient to have improvement of the symptoms of IBM including the ability to flex fingers and decreased anxiety of choking after 12 sessions of hyperbaric oxygen therapy.[10] Hyperbaric oxygen therapy also decreases pro-inflammatory cytokines such as IL-1, IL-6 and TNF-alpha. HBO has been shown to reduce inflammation and edema, promote angiogenesis and vasoconstriction, and mobilize bone marrow-derived stem progenitor cells.[11,12]

Platelet-rich plasma is a regenerative therapy that has been used in different applications and has shown the ability to repair damaged tissues, like skin ulcers and muscle and tendon injuries.[13] PRP is autologous plasma that is processed to provide a higher density platelet concentration. This higher concentration is injected into tissues with low healing potential, and it provides growth factors that act as a regenerative stimulus.[13] Hyperbaric oxygen therapy and PRP were initiated for synergist effects. There has been reported successful combination treatment seen in other conditions like osteomyelitis and diabetic wounds.[14]

This patient with inclusion body myositis experienced individual success with hyperbaric oxygen and PRP therapy. Hyperbaric oxygen therapy and PRP were used synergistically in conjunction with her traditional occupational and physical therapy. There continues to be no definitive effective treatment for IBM.[3]

CONCLUSION

Inclusion body myositis is an uncommon inflammatory myopathy and can be refractory to pharmacologic therapy and conventional treatments. This report highlights the novel use of these therapies as an adjunctive treatment for inclusion body myositis patients. In this report, the patient's symptoms and inclusion body myositis functional rating scale improved with hyperbaric oxygen therapy and platelet-rich plasma therapy. The patient met her treatment goals of maintaining and improving muscle strength, flexibility, and mobility. There continues to be no definitive effective treatment for inclusion body myositis, and additional research is warranted to further investigate the synergistic effects of hyperbaric oxygen therapy and platelet-rich plasma therapy on patients who are failing traditional therapies.

Funding: The Foundation for Optimal Health – 501 (c) (3), East Moriches, NY
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Dimachkie MM, Barohn RJ. Inclusion body myositis. Neurol clin. 2014;32(3):629-46.
2. Needham M, Mastaglia FL. Sporadic inclusion body myositis: A review of recent clinical advances and current approaches to diagnosis and treatment. Clin Neurophysiol. 2016;127(3):1764-73.
3. Rare National Organization for Rare Disorders. Sporadic inclusion body myositis, 2019. Available at: https://rarediseases.org/rare-diseases/sporadic-inclusion-body-myositis. Accessed on 20 December 2021.
4. Lilleker JB, Rietveld A, Pye SR. On behalf of all UKMYONET contributors, et al. Cytosolic 5′-nucleotidase 1A autoantibody profile and clinical characteristics in inclusion body myositis. Ann Rhem Dis. 2017;76:862-8.
5. Peng A, Koffman BM, Malley JD, Dalakas MC. Disease progression in sporadic inclusion body myositis: observations in 78 patients. Neurol. 2000;55(2):296-8.
6. Needham M, Mastaglia FL. Sporadic inclusion body myositis: A review of recent clinical advances and current approaches to diagnosis and treatment. Clin Neurophysiol. 2016;127(3):1764-73.

7. Naddaf E, Barohn RJ, Dimachkie MM. Inclusion Body Myositis: Update on pathogenesis and Treatment. Neurotherap. 2018;15(4):995-1005.
8. Morris G, Anderson G, Dean O. The glutathione system: a new drug target in neuroimmune disorders. Mol Neurobiol. 2014;50(3):1059-84.
9. Jackson CE, Barohn RJ, Gronseth G, Pandya S, Herbelin L. Muscle Study Group. Inclusion body myositis functional rating scale: a reliable and valid measure of disease severity. Mus Ner. 2008;37(4):473-6.
10. Pell M, Saththasivam P, Stephens PL, Mychaskiw G 2nd. Therapeutic effect of hyperbaric oxygen on inclusion body myositis. Undersea Hyperb Med. 2012;39(6):1111-4.
11. Sen S, Sen S. Therapeutic effects of hyperbaric oxygen: integrated review. Medical gas research. 2021;11(1):30-3.
12. Heyboer M, Milovanova TN, Wojcik S, Grant W, Chin M, Hardy KR et al. CD34+/CD45-dim stem cell mobilization by hyperbaric oxygen — Changes with oxygen dosage. Stem Cell Res. 2014;12(3):638-45.
13. Wu PI, Diaz R, Borg-Stein J. Platelet-rich plasma. Phys Med Rehabil Clin N Am. 2016;27(4):825-53.
14. Yümün G, Kahaman C, Kahaman N. Effects of hyperbaric oxygen therapy combined with platelet-rich plasma on diabetic wounds: an experimental rat model. Arch Med Sci. 2016;12(6):1370-76.

Cite this article as: Katz A, Thomson K, Morgan D. An emerging role of platelet-rich plasma and hyperbaric oxygen in the management of inclusion body myositis: a case report. Int J Adv Med 2022;9:756-9.