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A Review on Autoimmune and Inflammatory Myopathies-Idiopathic Inflammatory Myopathies

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Abstract

Idiopathic inflammatory myopathies (IIMs) are a group of lethal autoimmune muscle diseases. This study provides an update on the pathophysiology and changing importance of myositis-specific antibodies. The idiopathic inflammatory myopathies are subdivided into numerous clinically, histologically, and pathogenetically distinct groups, including dermatomyositis, polymyositis, inclusion body myositis (IBM), and autoimmune necrotizing myositis. IIMs have traditionally been categorised as polymyositis or dermatomyositis (NAM).The diagnosis of IIMs, which involves manual muscle testing, laboratory investigations, and non-invasive imaging, has become vital in order to categorise IIM subtypes and assess the severity of the ailment. From a time when glucocorticoid therapy was the only treatment option to the present, when immunoglobulin therapy, biologics like rituximab, and traditional steroid-sparing medications are all choices for treatment. Dermatomyositis, polymyositis, and immune-mediated necrotizing myopathy can all be treated successfully with immunosuppressive therapy; however, IBM, which is frequently resistant to currently available treatments, is not one of these conditions.

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INTRODUCTION

Idiopathic inflammatory myopathies (IIMs) are a class of autoimmune muscle illnesses that, if not treated well at a clinical evaluation early in the disease course, can cause severe morbidity and mortality. Based on clinical criteria established by Bohan and Peter in 1975 [1], this category of myopathies has long been categorised.

Yet, ongoing studies have shown the value of expanding myopathy subclassifications, frequently based on the presence of antibodies, which have further illuminated the pathogenesis and available therapies for inflammatory myopathies [2].

Inflammatory myopathies can frequently (11–44%) co-occur with other autoimmune diseases such as scleroderma, systemic lupus erythematosus (SLE), mixed connective tissue disease, rheumatoid arthritis, Sjogren's syndrome, polyarteritis nodosa, and sarcoidosis.

Diagnoses of inflammatory myositis that are less frequent include those for granulomatous myositis (also known as sarcoidosis), eosinophilic myositis, infectious myositis, and myositis linked to overlap syndromes.

Epidemiology

IIMs are thought to affect 4.27 to 7.89 instances per 100,000 people annually. With the exception of

inclusion body myositis, IIMs often start with proximal muscle weakness and can develop into systemic involvement of organ systems including the heart and lung, which has a high risk of morbidity. [3] Schiopu et al. investigated the impact of corticosteroids, methotrexate, and azathioprine on the progression of the illness in 160 individuals with a diagnosis of polymyositis/dermatomyositis. The findings of this study showed a 62% 10-year survival rate. Patients who got intravenous glucocorticoid therapy had a higher fatality rate than those who received methotrexate or azathioprine, which was thought to be because their diseases were diagnosed at a higher severity [3]. Several research teams have made additional efforts to further categorise the pathophysiology of IIMs in an effort to better identify and forecast which patient categories are more likely to develop interstitial lung disease (ILD), malignancy, and other IIM-related co-morbidities.

With the exception of inclusion body myositis, where the male to female ratio may be as high as 3:1, they afflict women more frequently than males. It's interesting to note that IIM is the most prevalent autoimmune myopathy in adults over 50, accounting for 16–28% of all myopathies, with an age-adjusted frequency of roughly 3.5/100,000 [4]. With age and ethnicity, the prevalence of inflammatory myopathies varies. According to certain research, African People have a greater incidence of polymyositis. Polymyositis typically first manifests in people who are older or in their late teens (mean age at onset in the 50s). There are two peaks for dermatomyositis: ages 5 to 15 and ages 45 to 65. The majority of people with IBM are over 50, while younger ones seldom get it [5]. Several research discuss the connection between inflammatory myopathies and cancer. According to several research, the prevalence of myopathies linked to cancer ranges from 4 to 42%, but overall, dermatomyositis patients are more likely to get cancer than polymyositis and IBM patients [6].

Classification

Idiopathic inflammatory myopathies have been divided into a number of categories. The initial Bohan and Peter criteria were created in 1975 [1] and were based on characteristics including symmetrical proximal muscle weakness, the typical dermatomyositis rash, myopathic alterations on electromyography, distinctive muscle biopsy, and elevated muscle enzymes. Before the discovery of muscle-specific autoantibodies, these standards were developed. Inclusion Body Myositis (IBM), which was not recognised until 1980, is not included in this classification. 6 Based on clinical observations, histology, and test results, an international session of myositis specialists recommended a second categorization in 2004. The following categories are included in the new categorization but myositis linked to other connective tissue diseases is excluded:

1. IBM
2. Polymyositis
3. Dermatomyositis
4. Amyopathic dermatomyositis
5. Non-specific myositis
6. Immune-mediated necrotizing myopathy

Inclusion Body Myositis

Because that CD8+T-cells are predominate on histopathology, inclusion body myositis may first appear with muscle biopsy pathology that is comparable to polymyositis. Vacuoles and staining for cytochrome-oxidase, p62, and congophilic amyloid deposits can distinguish polymyositis from inclusion body myositis [7]. The first histologic exam may be more consistent with the features found in polymyositis since these distinctive histopathologic findings for inclusion body myositis may not be evident in early disease activity. A more thorough clinical evaluation can result in a distinction, which is crucial because therapy choices for inclusion body myositis are limited in comparison to those for the other IIM subtypes [8]. With the quadriceps also being involved, inclusion body myositis primarily affects the distal muscles at first, including wrist extension, forearm mobility, and fine motor function of the hands. It may also impact the face and axial muscles, which is unusual for other IIMs. Also present is dysphagia. With a reported slowly progressing onset seen in people over the age of 50, this disease process is more sneaky. Men are the primary victims of the illness. Up to 10 times above the upper limit of normal serum CK levels are possible [9].

Polymyositis

The term "polymyositis" is becoming more widely used as a catch-all for IIMs that don't meet the precise requirements for classification into the other four subcategories or that test negative for myositis-specific antibodies. Subacute proximal symmetric muscular weakness is a common presenting symptom of individuals with dermatomyositis, although patients with polymyositis lack the usual rashes of dermatomyositis and have atypical results regarding muscle histology. The subacute active phase may

have serum CK levels that are up to 50 times over normal [9].

Dermatomyositis

Typical symptoms of dermatomyositis include proximal symmetric muscular weakening and related recognisable rashes. The shawl sign or V sign, Gottron papules, and heliotrope rashes across the eyelids with accompanying periorbital edoema are some of the distinctive skin rashes [10]. In the subacute active phase, creatine kinase (CK) levels may rise up to 50 times beyond the upper limit of normal. Amyopathic dermatomyositis, on the other hand, is a kind of dermatomyositis in which individuals have typical rashes but no muscular weakness. Individuals may also have typical muscle biopsy pathology and muscular weakness without rashes, a condition known as dermatomyositis sine dermatitis [11]. After three to five years of diagnosis, 15% of dermatomyositis patients diagnosed beyond the age of 40 have, or will have, a malignancy. The colon, ovarian, lung, pancreatic, and stomach cancers are the most often occurring malignancies connected to dermatomyositis.

Juvenile Dermatomyositis

With a frequency of 2-4 per million, juvenile-onset myositis is a fairly uncommon condition [12]. Juvenile dermatomyositis (JDM) is widely used as a catch-all term for all juvenile-onset myositis since the great majority of afflicted children also have cutaneous illness. There is still a lot of variation within the JDM subgroup, with different levels of chronicity, organ involvement, and long-term clinical prognosis. It is imperative to develop methods for more accurately classifying homogenous subgroups of JDM patients in order to facilitate diagnosis, provide prognostic information, and enable high-quality clinical studies of both novel and current treatments. In addition to histopathological correlations, recent investigations have demonstrated that autoantibodies may be detected in 60-95% of JDM patients and offer an additional degree of phenotypic refinement [13].

Anti-Synthetase Syndrome

Anti-synthetase syndrome stands out from the other subcategories because it is characterised by typical symmetric proximal muscle weakness and biopsy results that are compatible with dermatomyositis, but in 75% of patients, there is also a positive anti-Jo1 myositis-specific antibody. These symptoms, as well as arthritis, Raynaud's phenomenon, fever, and "mechanic's hands," are characteristic manifestations of anti-synthetase syndrome, with ILD occurring in 70% of patients. This diverse range of clinical manifestations is what gave rise to this distinct subclass of IIMs.

Necrotizing Autoimmune Myositis

20% of IIM patients are thought to fall under the subtype of necrotizing autoimmune myositis (NAM), which was previously likely categorised as polymyositis prior to the growing significance of histopathology. A considerable proximal symmetric weakness, which is frequently severe and incapacitating at first presentation, may appear acutely or subacutely with NAM. Compared to polymyositis/dermatomyositis, serum CK is frequently high, with values frequently exceeding 50 times the upper limit of normal. 3-hydroxy-3-methylglutaryl coenzyme, anti-signal recognition particle (anti-SRP), and myositis-specific antibodies Anti-HMGCR A reductase antibodies are unique to NAM. The development of anti-HMGCR antibodies was once thought to follow statin exposure; however, recently released material emphasises that individuals may not have taken statins but may still experience exposure to anti-HMGCR antibodies from other sources.

Ocular myositis

An uncommon inflammatory condition of the extraocular muscles (EOM) called ocular myositis makes for around 8% of all idiopathic EOM illnesses. flamboyant orbits. Eye movement might exacerbate the uncomfortable diplopia brought on by a single or several EOMs. Although corticosteroids continue to be the mainstay of therapy, radiotherapy and other steroid-sparing drugs are becoming more popular. Sometimes, ocular myositis may be a component of a more widespread inflammatory response. Hence, clinical, radiographic, and, where appropriate, histological data are used to classify IOI and the subtype of idiopathic orbital myositis [14].

Pathophysiology

Despite the fact that the cause and pathogenesis of idiopathic inflammatory myopathies are still unknown, a number of lines of research point to potential mechanisms by which certain environmental exposures in genetically vulnerable people may result in chronic immune activation and an immune attack on muscle and other involved tissues. Up-regulation of MHC class I expression and IL-1 alpha and beta are frequent immune activation pathways in muscle that result in autoantibody synthesis prior to the onset of clinical illness. Then, in polymyositis and IBM, myocyte-directed cytotoxic T-cell processes are dominant, but in dermatomyositis, complement-mediated endothelium damage resulting in CD4, B-cell, and dendritic cell infiltration is dominant in muscle tissue.

Hypoxia, the stimulation of the endoplasmic reticulum stress response, and the cleavage of autoantigens that results in the production of cytokines and chemokines are other potential disease pathways. Muscle regeneration, angiogenesis, healing, and, occasionally, fibrotic alterations are later processes [15].

A review of current pathophysiology supports the notion that antigens cause the C5b-9 macrophage-activating complex to become activated in people with dermatomyositis (MAC). When MAC is activated, it is secreted onto the surface of endothelial cells and identified as the antigenic target, which causes necrosis and ultimately results in capillary ischemia. The typical peri fascicular atrophy discovered by muscle biopsy is further triggered by this incident. Proinflammatory cytokines are also released by activated MAC, which causes the invasion of B cells, CD4+ T cells, and plasmacytoid dendritic cells [16].

It has been demonstrated that an antigen-driven response causes CD8+ T-cells to invade normally healthy muscle cells that express MHC-1, an abnormal protein for muscle cells, in individuals with polymyositis and inclusion body myositis. In this way, perforin and granzyme B are released by CD8+ T-cells, causing myonecrosis and ultimately damaging the endomysium. Increased TH17 synthesis and subsequent release of proinflammatory cytokines, such as IL-1, IL-6, and IL-15, cause an inflammatory environment in each of these three subcategories [11]. For the purpose of defining IIMs, [17] Dalakas&Hohfeld (2003) offered the following definitions based on the pathology and immunology of muscle biopsy: There are four types of polymyositis: (1) definite polymyositis, which exhibits inflammation with CD8+ T-cells/MHC-1 complex and no vacuoles; (2) probable polymyositis, which exhibits inflammation with MHC-1 but without CD8+ T-cells and no vacuoles; (3) dermatomyositis, which exhibits inflammation with peri fascicular, perimysial, or perivascular infiltrates; (4) While there are no rashes seen on a clinical examination, probable dermatomyositis has the characteristic muscle biopsy pattern seen in definitive dermatomyositis.

Diagnosis

The patient's medical history and the previously mentioned clinical findings point to an idiopathic myopathy diagnosis. Also, it's important to rule out additional conditions such as drug-induced myopathies, viral myopathies, hypothyroidism, myasthenia gravis, muscular dystrophies, rhabdomyolysis, sarcoidosis, and metabolic myopathies. The presence of certain antibodies, an increase in muscle enzymes, and specific EMG and MRI abnormalities can all help with the diagnosis. An open muscle biopsy is the only test that can conclusively determine the diagnosis of idiopathic inflammatory myopathies.

Muscle Enzymes:

While evaluating myopathies, serum muscle enzymes including creatine kinase (CK) and aldolase are frequently tested. While they are not specific for muscle disease, the enzymes lactate dehydrogenase (LDH), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) are frequently increased. Most dermatomyositis patients have elevated blood CK values, which can exceed 100 times the upper limit of normal. Serum CK levels may be normal in less than 10% of dermatomyositis patients, regardless of severity. Serum aldolase measurement can be useful since it occasionally occurs without a rise in CK levels. Similar to dermatomyositis, there is nearly always an elevation in CK levels in polymyositis, frequently more than 100 times the normal range. The blood CK levels in autoimmune necrotizing myopathy maybe ten times over the upper limits of normal. Although there is a positive clinical response to therapy, the blood levels of CK and aldolase do not correspond with the degree of muscular weakening. An elderly person's substantial weakness and near-normal muscle enzyme levels point to severe muscular atrophy or IBM [1].

Autoantibodies:

80% of individuals with polymyositis and dermatomyositis have antinuclear antibodies (ANA), which are detectable by traditional immunofluorescence techniques but are not particular to either disorder.

Anti-SS-A (anti-Ro), anti-SS-B (anti-La), anti-Smith, or anti-ribonucleoprotein (anti-RNP) antibody detection is highly suggestive of the diagnosis of myositis linked to overlap syndromes like MCTD or UCTD [18].

Myositis-specific Autoantibodies:

Myositis-specific autoantibodies refer to a number of subcategories of autoantibodies that are directed against cytoplasmic RNA synthetase, ribonucleoproteins, and certain nuclear antigens.

A serum ANA is not as selective as these antibodies, which are present in 30% of individuals with polymyositis and dermatomyositis.

Growing evidence points to a special function for myositis-specific autoantibodies in the pathogenesis of the idiopathic inflammatory myopathies. These antibodies fall into three main types [19].

Electromyography:

Electromyography (EMG), in addition to laboratory tests, is crucial in the assessment of patients with inflammatory myopathies. The standard triad below describes how the EMG demonstrates enhanced membrane irritability:

1. Spontaneous fibrillation and increased insertional activity
2. Abnormal myopathic polyphasic motor potentials with low amplitude and brief duration
3. Repeated complex discharges

In individuals with typical poly or dermatomyositis, a normal EMG is rare. Although they may help with the diagnosis, EMG anomalies are not exclusive to idiopathic inflammatory myopathies.

Magnetic Resonance Imaging:

An important method in the clinical assessment of individuals with inflammatory myopathies is magnetic resonance imaging (MRI). A myositis region with inflammatory alterations, edoema, muscular fibrosis, and calcification can be shown on an MRI. Due to the frequent limited or patchy inflammatory involvement of the muscles, it also aids in reducing sample error for a muscle biopsy. The ability to perform serial evaluations, which may be helpful in assessing a patient's response to therapy, is another benefit of MRI.

Muscle Biopsy:

Muscle biopsy is the only test that can conclusively determine if a patient has inflammatory myopathy, which affects the majority of myositis patients. A weak muscle should be selected for the biopsy based on physical assessment. Quadriceps or deltoid muscles are frequently used as biopsy sites. If at all feasible, the biopsy should be performed on a muscle from an EMG examination that was not pierced with a needle. It is not recommended to take a biopsy of the calf muscles since they frequently exhibit histological abnormalities and are not the proximal muscles where the illness is most pronounced. In situations when the original biopsy was insufficient to make the diagnosis or the EMG failed to locate the myopathy, MRI can also be used to determine the proper biopsy site. A more precise sample may be acquired and the muscle fibres are better maintained with an open biopsy than a closed needle biopsy. In one research, the total sensitivity of open muscle biopsy for the diagnosis of polymyositis and dermatomyositis was 83%. The sensitivity of identifying myositis can be increased by repeating the biopsy with MRI [20].

Treatment

For the treatment of IIMs, several immunosuppressive medications have been attempted as steroid-saving alternatives. Unlike the other IIMs, inclusion body myositis has not been demonstrated to react to immunosuppressive medications such glucocorticoids. In fact, intravenous immunoglobulin G is the only authorised pharmacological treatment for inclusion body myositis at this time. Except than inclusion body myositis, the remaining medications listed are choices for treating IIM subtypes.

IIMs continue to be mostly treated with glucocorticoids as the first line of defence. The suggested dose for patients with mild to moderate disease activity, as determined by the clinical exam results described above, is 1 mg/kg/day, not to exceed 80 mg daily. Pulse dose steroid treatment of 500–1000 mg daily followed by 1 mg/kg/day is used in patients with quickly progressing or severe illness as determined by clinical exam results, such as those patients with dysphagia and/or ILD and/or significant motor weakness. If improvements in the serum CK level or muscular strength are noticed, patients are frequently kept on steroid medication for at least 9 to 12 months with a gentle taper starting after 4 to 8 weeks of treatment [21].

Steroid-sparing treatment, which includes MTX and azathioprine (AZA), is known as first-line therapy. It is possible to start using MTX at a dose of up to 25 mg/week. Nevertheless, due to the possibility of MTX-induced pulmonary toxicity, vigilance must be urged in patients with ILD [8]. Muscle breakdown-related elevated serum SGOT/SGPT levels may make it more challenging to detect indicators of MTX intoxication. In individuals with ILD, AZA is frequently recommended, with a beginning dose of 50 mg/day and an increase in dose to a target of 1.5 mg/kg/day.

In IIM patients with severe or quickly progressing illness who lack tolerance or have a poor response to glucocorticoid treatment, intravenous immunoglobulin G (IVIG) therapy is being utilised more often. One of the few therapies recommended for inclusion body myositis, notably in those with oropharyngeal dysfunction, is IVIG. The usual method of administering IVIG treatment is via infusion at a dosage of 2 gm/kg/month [22].

Rituximab, a monoclonal antibody that targets CD-20 on the surface of pre-B cells, has also been utilised more often in cases with IIMs that have not responded to first-line treatments. Patients with overlap syndrome have also been treated with cyclophosphamide. Case reports indicated that the need for steroids was reduced, leading to clinical

improvement. Due to the presence of activated CD8+ T-cell in myositis-associated ILD, tacrolimus and cyclosporine have also been attempted since both medications operate to prevent T-lymphocyte proliferation and activation. Many case studies have reportedly used mycophenolate mofetil (MPF). Moreover, MPF is preferred for usage in individuals with overlap syndrome, connective tissue disease-associated ILD, or myositis-associated ILD [22]. TNF alpha suppression was suggested as a treatment option for IIMs since it was discovered that TNF was raised in the muscle tissue of IIMs. At this point, reports of TNF- blocking as a treatment for IIMs have not materialised, and TNF medication has been linked to additional occurrences of dermatomyositis and polymyositis [23].

CONCLUSION

IIMs have high morbidity and mortality rates, which can result in permanent impairment or even death. Myositis-specific antibodies and their function in IIMs are becoming more and more well-understood. These auto-antibodies are now being utilised more often to forecast IIM symptoms other than the conventional muscular weakness, such as ILD and the potential for cancer. By identifying these disorders in the earliest stages, ordering the initial tests such as muscle enzymes, EMG, or MRI, and sending the patients to specialists, primary care physicians play a crucial part in maximising the care of these patients. To rule out any concealed malignancy, these individuals frequently require appropriate cancer screening. Many immunologic agents are being tested in cases of resistant IIMs as we learn more about the aetiology of IIMs. As a result, these pathways are being targeted based on the disease's known aetiology. The management of these patients can be maximised and the negative effects of corticosteroids and other immunotherapies can be reduced by collaboration between the primary care physician and the specialist.

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