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Case Report

Inclusion body myositis in an older patient

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ABSTRACT

Inclusion body myositis (IBM) is a condition also referred to as sporadic IBM. It is a rare variant of a broader group of diseases described under the banner of inflammatory myositis. In general, myositis/myositides tend to present with a varying cluster of symptoms such as muscular weakness, aches and/or pain. Myositis has been linked to multiple causes or triggers, but generally share the common feature (present to a variable extent) of inflammatory changes within skeletal muscle tissue. In addition, some associations (e.g. Sjogren's disease and systemic lupus erythematosus – SLE) have been reported for IBM. Some medication classes (e.g. statins and fibrates) may present a risk to certain individuals developing drug-associated myopathies. IBM can be challenging to diagnose, and may be mistaken for many other causes of muscle weakness e.g. polymyositis. Furthermore, IBM tends to affect muscles asymmetrically, and runs a typically progressive and chronic course. Consequently, over time IBM may result in significant functional impairment, activity limitation, and participation restriction. This case report describes an older woman with a clinical diagnosis of probable IBM complicated with oesophageal dysmotility. She presented to hospital with progressive dysphagia, breathlessness, and a productive cough for which treatment was started for aspiration pneumonia. The report considers some broader 'principles of management' of a chronic myositis. In the current absence of a definitive curative treatment, we also discuss some realistic and practical pharmacological treatment options that may be used for the selective care of patients presenting with an incurable chronic myositis such as IBM.

Keywords: Chronic disease, Inclusion body myositis, Myopathy, Myositis, Older adult, Sporadic myositis

INTRODUCTION

Myositis is a condition characterised by inflammatory changes within muscle tissue, and arising from multiple possible causes, and/or triggers.[1-5] The condition may present with a myriad of symptoms, some of which include muscle weakness, aching and/or pain.[6]

Inclusion body myositis (IBM) or sporadic IBM is a rarer subtype of myositis that is often classified under the spectrum of 'idiopathic' inflammatory myopathies or myositis.[1,3,7,8] IBM is described as having a male > female prevalence.[8] IBM can be a diagnostic challenge, and often subject to a delayed diagnosis.[8] IBM generally runs a chronic and progressive course, with asymmetric muscle affectation.[8] The latter in turn can cause significant functional impairment, adverse impact upon quality of life, as well as increased morbidity and mortality.

Given the polysymptomatic nature, people with myositis (in general) and IBM (more specifically) may variably present to clinicians across a wide range of specialties, and also to primary, secondary or tertiary care practitioners. IBM may also be confused with polymyositis, and/or a host of other differential diagnoses, which may further impact on the adopted approach to assessment and management.[6,7]

In this case report, we describe an older woman with probable IBM admitted to hospital with a two-week history of progressive dysphagia, intermittent chest tightness, dyspnoea, and a productive cough. She had

oesophageal dysmotility, resulting in dysphagia and sadly complicated by aspiration pneumonia. In our discussion, we reflect on some of the broader 'principles of management' of what is essentially a chronic condition/disease, as exemplified from the perspective of an affected older patient. In addition, we consider a range of (practical) pharmacological management options that may be used (selectively) during the care of patients presenting with chronic myopathies such as IBM.

CASE REPORT

An 83-year-old woman was admitted to hospital with a two-week history of progressive dysphagia. She also described intermittent feelings of tightness in her chest, dyspnoea, and a cough productive of white phlegm.

Pre-admission, she lived in a care home setting, and required assistance to maintain her activities of daily living. Her mobility indoors was aided with a Zimmer frame, and outdoors with a wheelchair. She was teetotal and a non-smoker. She weighed 53.2 kg, height 1.60 metres, and body mass index (BMI) 20.7.

Her extensive medical history included cardiovascular issues of congenital complete heart block and childhood rheumatic fever, necessitating the insertion of a permanent pacemaker implantation later in life. In addition, she had hypertension, atrial fibrillation, moderate left ventricular systolic dysfunction and an ischaemic posterior circulation stroke. From a gastroenterology perspective, she had oesophageal dysmotility confirmed on manometry, and had undergone a laparoscopic cholecystectomy for gallstones. She was further noted to have gastritis and oesophagitis on endoscopy, with the presence of hyperplastic polyps on gastric biopsies. Other notable issues were lumbar spinal stenosis and osteopenia. Five years prior to this acute presentation, she had undergone a muscle biopsy for unexplained muscle weakness and pain. The histopathologist had considered a differential diagnosis of either pyomyositis or IBM. A retrospective review of her clinical records indicated that the IBM had initially presented as progressive muscular weakness which impacted on her mobility. She had subsequently developed chronic dysphagia. There was no history of thyroid dysfunction, diabetes mellitus, rheumatologic disorders, autoimmune disease or malignancy.

Her medications (all oral) on admission were: codeine suspension 15mg four times daily, diazepam tablets 5 mg as required, lactulose 10 ml twice daily, lansoprazole dispersible gastro-resistant tablets 20mg daily, paracetamol suspension 1 gram four times daily, and senna suspension 15mg at night. She had a documented allergy (rash) to penicillin.

On admission, her Glasgow coma scale was 15/15, and observations noted a temperature of 36.4°C, blood pressure 138/69 mmHg, pulse 76 bpm, respiratory rate 24/min. Her oxygen saturations were initially very low (75-80% range) when breathing air, improving to 94% on supplemental oxygen via nasal prongs at a flow rate of 2 l/min.

Her clinical (respiratory) findings and plain radiological (chest X-ray) features were indicative of a pneumonia. Except for dehydration and the noted presence of multi-focal muscle weakness, her other general and systemic examinations were unremarkable.

Her admission blood workup showed a normal haemoglobin and platelet count. She had a neutrophil-predominant leucocytosis of 14.4×10^9/l (4–11), suggestive of a probable bacterial infection. C-reactive peptide was markedly elevated at 271 mg/l (0–5). Renal function tests suggested dehydration with serum urea 11.8 mmol/l (2.5–6.6), creatinine 54 mmol/l (50–98), and a normal estimated glomerular filtration rate (MDRD) for age of >60 ml/min/1.73 m2. Liver function tests were normal except for serum albumin 29 g/l (36–47). Serum B12, folate and ferritin levels were normal, as were thyroid function tests. Serum electrolytes noted potassium 3.0 mmol/l (3.6–5), magnesium 0.62 mmol/l (0.7–1.0), phosphate 0.62 mmol/l (0.8–1.4), but normal serum (corrected) calcium. Random serum glucose was normal. Arterial blood gases showed mild hypoxaemia and mild hypercapnia. Two serial highly sensitive troponin I assays (done as part of the assessments of her chest tightness) did not show a significant rise over a 3-hour interval.

A resting 12-lead electrocardiogram (ECG) showed an unremarkable trace in sinus rhythm.

Microbiology (sputum) bacterial cultures grew respiratory commensals only. She was negative for atypical pathogens, including Mycoplasma pneumoniae. Virology (throat swab) was PCR positive for rhinovirus, but negative for a host of other viruses (influenza A and B; parainfluenza types 1, 2, 3 and 4; coronaviridae; metapneumovirus; and human bocavirus – a type of parvovirus).

Her clinical records also showed prior detailed investigations into the cause of the muscle weakness and myositis. Over time, she had mildly elevated serum creatine kinase levels ranging between 17–451 UI/l (30–135). Serum 25-OH vitamin D level was 37 nmol/l (deficiency ≤24; insufficiency 25–49; normal ≥50).

Extensive immunological tests were all negative (with the range of immunology including ENA and ANA panels, cyclic citrullinated peptide - CCP antibodies, acetylcholine receptor - AChR antibodies, smooth muscle antibodies - SMA, and anti-thyroid peroxidase antibodies).

Prior advanced imaging (computerised tomography - CT) scans of her abdomen and pelvis had not identified the presence of a malignancy.

Results of the previous skeletal muscle biopsies are summarised in Table 1.

Table 1: Excerpts from detailed histology report of previous skeletal muscle biopsies.

S. no.	Histology reports
	Notes on the skeletal muscle biopsy samples
1	A range of muscle fibre shapes and sizes. Scattered atrophic fibres which are rounded, and some forming small groups
2	The ATPase preparations showed a predominance of type I fibres, although the small groups appeared to be a mixture of both type I and type II fibre
3	Presence of mild increase in connective tissue separating fibres highlighted by the trichrome stain and adipose tissue infiltration into the skeletal muscle fascicles
4	Prominent inflammation mostly surrounding individual skeletal muscle fibres and associated with fibre necrosis. In one area, the appearances were almost granulomatous, although these likely represent several necrotic fibres adjacent to each other
5	Very occasional vacuoles seen within skeletal muscle fibres, but none had a definite rimmed vacuole appearance. Occasional necrotic fibres showed some peripheral vacuolation, but these were not representative of 'typical' inclusion bodies
6	No ragged red fibres seen on the trichrome stain, and there was a normal distribution of lipid and glycogen within fibres
7	The NADH preparation showed a number of moth-eaten fibres, but no other significant abnormality
8	Immunohistochemistry showed the inflammatory infiltrate to be predominantly T-lymphocytic (CD3 positive) with occasional B-lymphocytes (CD20 positive). CD68 highlighted a marked up-regulation in macrophages particularly in relation to necrotic fibres. MHC class I was noted to be up-regulated throughout the biopsy
9	Tau, beta-amyloid and ubiquitin showed no significant staining within fibres
	Summary of consultant histopathologist's comments
1	These skeletal muscle biopsies show an inflammatory myopathy
2	The changes are longstanding with evidence of fibrosis and lipid infiltration of the muscle fascicles
3	Although focally the inflammation appears granulomatous, the overall appearances are not thought to be a granulomatous myositis
4	Many of the histological appearances are typical of a polymyositis, although inclusion body myopathy is a possibility that cannot be excluded from these biopsy findings

Following clinical non-response to a treatment trial with oral dexamethasone, a consultant neurologist refined/revised her working diagnosis to be more likely that of a case of sporadic IBM, i.e. rather than polymyositis. The clinical rationale was that IBM is often misdiagnosed as polymyositis (both clinically and on potentially challenging to interpret muscle histology samples). Some other practical distinctions (which were relevant to the index case) are that polymyositis-related muscle weakness often develops over 'weeks to months', and tends to be steroid responsive. This contrasts with the more typical pattern of IBM-related muscle weakness (which this patient exhibited) of her condition being: of slower onset of 'months to years'; having a tendency to be progressive in nature, and typically not being steroid responsive.

Therefore, given her clinical history of probable IBM, oesophageal dysmotility, progressive dysphagia, and this acute respiratory presentation, she was diagnosed with a complication of aspiration pneumonia and an associated rhinovirus infection. She was treated with controlled supplemental oxygen (for hypoxia in the context of a type 2 respiratory failure). She received intravenous (IV) antibiotics (co-trimoxazole and metronidazole) as empirical cover for aspiration pneumonia (with the antibiotic choices modified by the context of a known penicillin allergy in the form of a rash). In addition, she received IV fluids (for dehydration), as well as selective oral and parenteral correction of electrolyte imbalances (guided by the earlier stated blood results of hypokalaemia, hypomagnesaemia, and hypophosphataemia). Oral suspension paracetamol was continued (as analgesia and antipyretic). As the occurrence of Rhinovirus infections in adults often produces milder symptomatology, the treatment for this was mainly supportive, and without the use of specific anti-viral agents. Furthermore, she was reviewed by speech and language therapists (SaLT), with recommendations made for a modified texture diet (food and drink) to reduce aspiration risks.

Sadly, despite the active treatment measures instituted, the patient's condition steadily deteriorated with an increased oxygen requirement over the subsequent week. In consultation with her family, and duly noting her previously expressed wishes (an active 'do not attempt cardio-pulmonary resuscitation' – DNACPR instruction), her treatment was modified to palliative care measures. She subsequently died ten days into the acute admission. Her medical certificate of cause of death (MCCD), recorded the sequence and cause of death as complications linked to the evolving medical polymorbidity: 'inclusion body myositis'

causing 'oesophageal dysmotility', in turn causing 'dysphagia', and finally resulting in death from 'aspiration pneumonia'

DISCUSSION

Myositis

Myositis is a disease condition typically characterised by a spectrum of inflammatory changes within muscle tissue.[1,2] The condition may arise from multiple potential causes, and/or in relation to predisposing conditions or triggers.[1-5] In general, myositis often presents with a cluster of symptoms: including muscle weakness, as well as muscle aches and/or pain.

Inclusion body myositis – IBM

IBM or sporadic IBM falls under the spectrum of idiopathic inflammatory myositis/myopathy.[1,3,8,9] It is usually seen in the over 50-year adult population, and tends to affect males more than females.[3,8,7,10-12] The prevalence of IBM is varies from region to region e.g. due to factors like a tendency for the diagnosis to be missed or delayed, lack of awareness of this condition among generalist healthcare professionals, evolving diagnostic criteria, as well as incremental advances in molecular medicine and histopathology.[8]

In IBM, there is a gradual progression of (often initially) painless muscle weakness, which is of asymmetric nature, and leading to significant functional impairment/reduction.[8,12,13] Affected adults typically present with weakness of the following muscle groups: knee extensors (e.g. with individuals having difficulty standing or walking, thereby leading to falls); long finger flexors usually on the non-dominant side (e.g. presenting with weakness of grip strength or loss of dexterity leading to the dropping objects); ankle dorsiflexors (e.g. presentations with tripping due to foot drop); and/or also cricopharyngeal muscle dysfunction (e.g. resulting in dysphagia, albeit less often as a primary symptom, but in the later stages potentially resulting in weight loss and aspiration pneumonia).[8,9,12]

The diagnostic criteria for IBM require: the presence of an asymmetric pattern of clinical weakness e.g. either knee extensors or finger flexor; muscle biopsy confirming the presence of rimmed vacuoles (although this may be absent in up to 20% of individuals), or noting the invasion by T-cells of non-necrotic muscle fibres and the presence of endomysial inflammation.[3,8,12]

The presence of anti-cytosolic 5'-nucleotidase 1A (cN1A) autoantibody (described/discovered in year 2013) can also be a helpful aid in establishing the diagnosis of sIBM i.e. when used alongside the criteria above.[3,8] A limitation is that this named autoantibody is thought to be a 'myositis-associated' antibody, rather than it necessarily being a 'myositis-specific' antibody.[3,8,12] This is because it has also been observed in other conditions such as Sjögren syndrome and systemic lupus erythematosus (SLE).[3,8,12]

The use of magnetic resonance imaging (MRI) in sIBM can also be used to support the diagnostic formulation i.e. when used as a complementary investigative tool in addition to the clinical history, physical examination, muscle biopsy/histopathology, and the anti-cN1A antibody testing.[12] The MRI may show large areas of fibro-fatty change associated with muscle atrophy, and especially affecting the following muscle groups: the quadriceps and the flexor digitorum profundus.[13]

The pathogenesis of the muscle inflammation in IBM is thought to involve two components. Firstly, by a degenerative process which is demonstrable by the presence of rimmed vacuoles, protein aggregates, myonuclear degeneration and mitochondrial pathology.8 Secondly, by an autoimmune process as highlighted by the presence of T-cells, myeloid dendritic cells, plasma cells and macrophages.[8]

Some differential diagnoses of IBM

In practice, IBM can be inaccurately or incorrectly diagnosed as polymyositis (e.g. if the presence of rimmed vacuoles is missed on muscle biopsy samples/specimens), or it may be clinically misdiagnosed as being a form of motor neuron disease (MND) (e.g. as may be the case without muscle biopsy/biopsies).[7,11,13,14]

In the index case, polymyositis was deemed (clinically) unlikely due to the evolutionary timelines of her symptoms, and on account of the poor clinical response to a trial of steroid therapy.

Some other relevant differential diagnoses that were considered, but deemed unlikely in this case, as guided by clinical and/or other histological grounds were: MND, a drug-associated myopathy, myasthenia gravis, Lambert-Eaton Myasthenic syndrome (LEMS), diabetic amyotrophy, thyrotoxic myopathy, post-viral myocarditis, muscular dystrophies (e.g. Duchenne's), myotonic dystrophy, neuromyelitis optica spectrum disorder (NMOSD), other congenital or hereditary causes e.g. mitochondrial myopathies, glycogen storage disorders, and channelopathies (e.g. hypokalemic periodic paralysis).[1,2,8,9,14-18]

General principles of management of IBM

Some prospective treatment options have been explored (e.g. steroids, other immunosuppressants and/or other immunomodulators, and human monoclonal antibodies), but at this time, in the absence of a definitive cure or curative treatments, the management of IBM is often re-directed towards supportive measures and symptom-alleviation.[6,19] The range of symptoms may vary from patient to patient, and the clinical symptomatology (and/or severity) might also evolve over time as the condition

progresses.[6-9] Both in the pre-diagnosis and post-diagnosis phases, the affected individual is often required to acquire or adopt some self-management skills, measures and strategies relevant to chronic disease management.

Other persons that may be involved in an individual patient's care include family members and carers. Furthermore, dependent upon local availability the acute and chronic-phase management of patients with IBM may involve a range of professionals working as a multi-disciplinary team (MDT). These could potentially include: specialist physicians e.g., neurologists, neurophysiologists or rheumatologists for assessments of presentations with muscle weakness and pain. Gastroenterologists may be consulted for dysphagia. Acute, general, and geriatric medicine physicians may see patients on account of pain, mobility problems, falls, and continence issues. General practitioners, psychiatrists, psychologists, counsellors, and/or other mental health practitioners may be approached for affective disorders, mood changes, and emotional support.

Dependent upon need, other key members of the MDT may include nursing care practitioners. Allied health professionals (AHPs) may include physiotherapists for muscle strength, gait and balance, transfers, mobility including step/stair practice sessions, falls risk assessment, confidence-building, developing their exercise tolerance, and also occasionally for chest physiotherapy (e.g. mucus plugging). Other potential AHP input include: occupational therapists (OTs) for functional assessments and individualised adaptations, including falls prevention, and the use of assistive technologies. Additionally, SaLT may support swallowing assessments and offer recommendations on appropriate modifications to textures of food or drinks, as well as for speech assessments and recommendations on compensatory strategies and/or speech-augmenting assistive devices and technologies. Dietitians may also be helpful for personalised nutritional assessments and/or guidance on dietary supplementation.

Furthermore, other professionals that may support patient's care include: social workers for review and guidance on available or accessible community or voluntary support services. Clinical pharmacists may also offer crucial advice on potential adverse drug reactions and/or interactions that may be relevant to potentially worsening myopathy. As dysphagia progresses, pharmacists may also helpfully advice on alternative or suitable drug formulations e.g. oral suspensions, oral dispersible tablets, including advise on tablets that may or may not be suitably crushed like modified release preparations.

Some basic principles and general options for the pharmacological management of IBM

As highlighted earlier, the range and severity of an individual patient's symptoms may vary, and particularly as the condition itself might deteriorate over time. Therefore, in the context of a potentially incurable chronic disease condition, the pharmacological management options for IBM would generally revolve around the amelioration of symptoms, and/or treatment of potential complications of the condition.[6,19]

Such pharmacological options include: analgesics (pain and discomfort) and steroids (anti-inflammatory role, potentially to ameliorate low energy and as a short-term appetite stimulant). Others may require gastrointestinal prokinetics and/or stimulant and softener laxatives (oesophageal, gastric and intestinal dysmotility; and chronic constipation), or muscle relaxants and anti-spasmodic agents (muscle pain, stiffness, cramps or spasms).[20]

In addition, patients requiring long-term steroid therapy or multiple/recurring courses of oral steroids may benefit from bone health assessments/evaluations to allow for a clinical decision on whether or not it might be appropriate to consider additional bone-protective agents (e.g. calcium and/or vitamin D supplements, and bisphosphonates).[20]

Other potential pharmacological treatment measures that may need to be considered (on an individual patient basis) include mood-affecting treatments (e.g. anti-depressants and anxiolytic agents). Antibiotics may be required for treatment of specific infections (e.g. aspiration pneumonia from dysphagia, hypostatic pneumonias from reduced mobility, urinary tract infections from urinary bladder muscle hypotonia and potential risks of urinary retention). Some patients may also derive symptomatic benefit from the selective use of mucolytic agents to relieve thick/viscid phlegm. The latter may be especially relevant if they develop a weakened cough reflex (e.g. oral suspension or tablets carbocysteine, or nebulised saline solutions to aid expectoration).[20]

It is also important to highlight that another important general principle or facet of management is the need to consider stopping, avoiding or reducing doses of certain groups of medications that could worsen the risks of myositis. A non-exhaustive list includes statins, fibrates, macrolide antibiotics, D-penicillamine, protease inhibitors, colchicine, chloroquine, and amphetamines. Although steroids (e.g. dexamethasone) may be employed in some cases as treatment trials to aid distinguishing between polymyositis versus IBM (as was the case in this patient), it is equally important for clinicians to recognise that long term steroid therapy can itself be a cause for (steroid-induced or steroid-associated) proximal myopathies. Steroid-associated diabetes mellitus could further pre-dispose to longer term problems like diabetic amyotrophy. Similarly, excessive alcohol consumption may be associated with myopathic changes, and counselling on alcohol reduction or cessation may be relevant to some individuals.[20]

Based on the general measures described above, it is plausible that over time, patients may acquire an increasing list of polypharmacy for the myriad of potential symptoms.

Consequently, it is important for clinicians to undertake periodic reviews of their patients' medications (whether prescribed or over-the-counter options) with a view to identifying any possible side-effects, ADRs and/or risk of drug interactions.

Equally, as patients may evolve into the palliative phase of the condition (or due to other concurrent conditions that may a have life-limiting impact), clinicians should remain proactive in rationalising and reviewing medications that are no longer required or no longer beneficial to patients. This serves the dual purpose of reducing the burden of inappropriate polypharmacy, and also reducing the risks of potential side-effects.

The index patient received supplemental oxygenation, analgesics (oral suspensions of paracetamol and codeine), and gastromotility/prokinetic agents (oral suspension senna). She also received antibiotics for aspiration pneumonia (IV co-trimoxazole and metronidazole; in the presence of a documented penicillin allergy), and muscle relaxants (oral diazepam). She had previously received steroid therapy (short term oral dexamethasone 4 mg daily for 2 weeks) without significant clinical benefit. She received gastroprotective therapy (oral dispersible lansoprazole). Nutritional supplementation and dietary texture modifications were also provided. Unfortunately, the advanced status of the patient's IBM resulted in an eventual transition from the initial unsuccessful attempts at active management to palliative care measures, and she subsequently died peacefully in hospital.

CONCLUSION

In this report, we describe the case of an older female patient with probable IBM who developed acute clinical features (symptoms and signs) and plain radiological evidence of an aspiration pneumonia. This was in the context of IBM-associated oesophageal dysmotility and progressive dysphagia.

The report reviews some general strategies that an MDT may consider in planning for the practical and personalised care of patients with this chronic/long term condition. This report describes measures in the setting of a specified myopathy (IBM). However, it is possible that clinicians may identify the potential value of considering and/or adapting similar multi-faceted approaches to the assessment and care of patients with other forms of chronic myopathies. These generic strategies may also prove relevant to, and/or impact upon patients' presenting with a range of other chronic and/or potentially debilitating inflammatory, rheumatological, auto-immune/immunological, or paraneoplastic muscle disorders.

The pharmacological measures described are not of definitive curative intent for IBM or other chronic myopathies. However, they might offer the prospect of an improved symptom profile to selected patients. This in turn may enhance their overall quality of life, i.e. while living with what can be a fairly disabling (both activity limiting and participation restricting) long term condition.

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