

Inclusion body myositis: clinical features and pathogenesis

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Abstract | Inclusion body myositis (IBM) is often viewed as an enigmatic disease with uncertain pathogenic mechanisms and confusion around diagnosis, classification and prospects for treatment. Its clinical features (finger flexor and quadriceps weakness) and pathological features (invasion of myofibres by cytotoxic T cells) are unique among muscle diseases. Although IBM T cell autoimmunity has long been recognized, enormous attention has been focused for decades on several biomarkers of myofibre protein aggregates, which are present in <1% of myofibres in patients with IBM. This focus has given rise, together with the relative treatment refractoriness of IBM, to a competing view that IBM is not an autoimmune disease. Findings from the past decade that implicate autoimmunity in IBM include the identification of a circulating autoantibody (anti-cN1A); the absence of any statistically significant genetic risk factor other than the common autoimmune disease 8.1 MHC haplotype in whole-genome sequencing studies; the presence of a marked cytotoxic T cell signature in gene expression studies; and the identification in muscle and blood of large populations of clonal highly differentiated cytotoxic CD8⁺ T cells that are resistant to many immunotherapies. Mounting evidence that IBM is an autoimmune T cell-mediated disease provides hope that future therapies directed towards depleting these cells could be effective.

Inclusion body myositis (IBM) is a slowly progressive skeletal muscle disease with unique clinical and pathological features (including finger flexor and quadriceps weakness and the presence of infiltrating cytotoxic T cells in muscle). In patients with IBM, the onset of symptoms of muscle weakness typically occurs between 45 and 70 years of age, and progression is insidious. The combination of slow progression towards disability and the lack of any reliable treatment to reverse the disease course results in a substantial unmet medical need.

IBM lies within the category of inflammatory myopathies. This category started with the single member, polymyositis, in 1887 (REF¹) and has since undergone repeated subdivision (FIG. 1). Dermatomyositis was recognized as a separate entity early in 1891 (REF^{2,3}), but it took almost a century before the next refinement, IBM, was adequately recognized in 1978 (REF⁴). Immune-mediated necrotizing myopathy (IMNM) was distinguished in 1991 (REF⁵). Advances continue to reduce the remaining category of polymyositis⁶, splitting off non-specific myositis⁷ (or unclassified myositis⁸) and virtually eliminating the term polymyositis from what once constituted the entire category of inflammatory myopathies. The generally accepted scheme currently consists of five members (polymyositis, dermatomyositis, IBM, IMNM and non-specific myositis)^{9,10} plus other rare entities (such as granulomatous myositis

and macrophagic myofasciitis). Further subdivision of dermatomyositis into distinct syndromes associated with specific autoantibodies is currently an active area of research¹¹.

Many excellent general IBM reviews have been published over the past 5 years^{12–15}, and some have focused specifically on the pathogenesis^{16,17}. This Review discusses IBM generally, with a focus on some of the more confusing areas around nomenclature and clinical features, and addresses key questions regarding its pathogenesis.

History of IBM

A historical perspective provides some insight into what has shaped our current understanding of IBM (FIG. 1). It was first viewed as a form of polymyositis resulting from chronic viral infection of muscle in 1967 (REF¹⁸), leading to an ongoing line of research directed towards the identification of a persistent muscle viral infection^{19–26}. The name inclusion body myositis is derived from a 1971 single case report entitled 'Inclusion body myositis'²⁷ that described a 26-year-old patient with a limb-girdle pattern of weakness and quadriceps sparing with onset at age 18 years (which is not the disease we now call IBM).

The first IBM clinical case series was published in 1978 and described patients with a distinct form of inflammatory myopathy characterized by prominent

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The Official Patients Sourcebook On Inclusion Body Myositis Pb 2002

JE Gale



The Official Patients Sourcebook On Inclusion Body Myositis Pb 2002:

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