



Complete Summary

GUIDELINE TITLE

EFNS guideline on diagnosis and management of limb girdle muscular dystrophies.

BIBLIOGRAPHIC SOURCE(S)

Norwood F, de Visser M, Eymard B, Lochmuller H, Bushby K, EFNS Guideline Task Force. EFNS guideline on diagnosis and management of limb girdle muscular dystrophies. *Eur J Neurol* 2007 Dec;14(12):1305-12. [28 references] [PubMed](#)

GUIDELINE STATUS

This is the current release of the guideline.

Because of the rate of advance in this group of conditions, it is suggested that these guidelines are reviewed after 2 years.

COMPLETE SUMMARY CONTENT

SCOPE
METHODOLOGY - including Rating Scheme and Cost Analysis
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SCOPE

DISEASE/CONDITION(S)

Limb girdle muscular dystrophy (LGMD)

- Autosomal dominant disease
- Recessive disease

GUIDELINE CATEGORY

Counseling
Diagnosis
Evaluation
Management

CLINICAL SPECIALTY

Cardiology
Family Practice
Internal Medicine
Medical Genetics
Neurology
Pulmonary Medicine

INTENDED USERS

Clinical Laboratory Personnel
Physicians
Respiratory Care Practitioners

GUIDELINE OBJECTIVE(S)

- To provide guidelines for the best practice management of the limb girdle muscular dystrophies based on the current state of clinical and scientific knowledge in the published literature
- To provide an approach to the diagnosis and monitoring of the limb girdle dystrophies in a manner accessible to general neurologists

TARGET POPULATION

Patients with limb girdle muscular dystrophies (LGMD)

INTERVENTIONS AND PRACTICES CONSIDERED

Diagnosis/Evaluation

1. Clinical assessment including medical history, family history, and assessment of clinical features
2. Laboratory tests including serum creatine kinase, muscle biopsy, immunohistochemistry and immunoblotting
3. Muscle imaging

Management

1. Respiratory management including monitoring of respiratory function, prompt treatment of respiratory infections, annual influenza vaccination, liaison with respiratory physician
2. Cardiac management (permanent pacing or implantable defibrillator, angiotensin-converting enzyme inhibitors, anticoagulation)
3. Gentle exercise and avoidance of prolonged immobility
4. Genetic counseling
5. Corticosteroids

MAJOR OUTCOMES CONSIDERED

Not stated

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

Search Strategy

The following search protocols were employed with relevant keywords: MEDLINE for original papers and review articles (1985–2005), Cochrane database, American Academy of Neurology (AAN) and European Federation of Neurological Sciences (EFNS) practice parameters or management guidelines, EMBASE, patient organizations, previous guidelines.

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Evidence Classification Scheme for a Diagnostic Measure

Class I: A prospective study in a broad spectrum of persons with the suspected condition, using a "gold standard" for case definition, where the test is applied in a blinded evaluation, and enabling the assessment of appropriate tests of diagnostic accuracy

Class II: A prospective study of a narrow spectrum of persons with the suspected condition, or a well-designed retrospective study of a broad spectrum of persons with an established condition (by "gold standard") compared to a broad spectrum of controls, where test is applied in a blinded evaluation, and enabling the assessment of appropriate tests of diagnostic accuracy

Class III: Evidence provided by a retrospective study where either persons with the established condition or controls are of a narrow spectrum, and where test is applied in a blinded evaluation

Class IV: Any design where test is not applied in blinded evaluation OR evidence provided by expert opinion alone or in descriptive case series (without controls)

Evidence Classification Scheme for a Therapeutic Intervention

Class I: An adequately powered prospective, randomized, controlled clinical trial with masked outcome assessment in a representative population or an adequately powered systematic review of prospective randomized controlled clinical trials with masked outcome assessment in representative populations. The following are required:

- a. Randomization concealment
- b. Primary outcome(s) is/are clearly defined
- c. Exclusion/inclusion criteria are clearly defined
- d. Adequate accounting for dropouts and crossovers with numbers sufficiently low to have minimal potential for bias
- e. Relevant baseline characteristics are presented and substantially equivalent among treatment groups or there is appropriate statistical adjustment for differences

Class II: Prospective matched-group cohort study in a representative population with masked outcome assessment that meets a–e above or a randomized, controlled trial in a representative population that lacks one criteria a–e

Class III: All other controlled trials (including well-defined natural history controls or patients serving as own controls) in a representative population, where outcome assessment is independent of patient treatment

Class IV: Evidence from uncontrolled studies, case series, case reports, or expert opinion

METHODS USED TO ANALYZE THE EVIDENCE

Review of Published Meta-Analyses
Systematic Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

The results of the literature review were evaluated by members of the task force; only those studies specific to limb girdle muscular dystrophies (LGMD) or the subtypes have been included. Older studies (pre-1985) include cases of 'limb girdle dystrophy' but without accurate molecular diagnosis it is not possible to extract reliable data from these, and so they have been excluded. All the evidence was categorized as class IV.

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

Not stated

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Rating of Recommendations for a Diagnostic Measure

Level A rating (established as useful/predictive or not useful/predictive) requires at least one convincing class I study or at least two consistent, convincing class II studies.

Level B rating (established as probably useful/predictive or not useful/predictive) requires at least one convincing class II study or overwhelming class III evidence.

Level C rating (established as possibly useful/predictive or not useful/predictive) requires at least two convincing class III studies.

Rating of Recommendations for a Therapeutic Intervention

Level A rating (established as effective, ineffective, or harmful) requires at least one convincing class I study or at least two consistent, convincing class II studies.

Level B rating (probably effective, ineffective, or harmful) requires at least one convincing class II study or overwhelming class III evidence.

Level C rating (possibly effective, ineffective, or harmful) requires at least two convincing class III studies.

Good Practice Points Where there was a lack of evidence but consensus was clear, the Task Force has stated their opinion as good practice points.

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

The guidelines were validated according to the European Federation of Neurological Societies (EFNS) criteria (see the "Availability of Companion Documents" field in this summary).

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

The levels of evidence (class I-IV) supporting the recommendations and ratings of recommendations (A-C, **Good Practice Points**) are defined at the end of the "Major Recommendations" field

All the evidence was categorized as Class IV.

Expert Consensus Recommendations for Management of Limb Girdle Muscular Dystrophies (LGMD)

Aspects of Care – Diagnosis

Clinical Assessment

General Principles

Thorough clinical assessment provides the basis for directing further investigation. Neonatal course, timing of developmental motor milestones and ability to rise from the floor/presence of Gower's manoeuvre may all be of relevance. The ability to run, hop and jump and sporting ability may be significantly affected in childhood or may be normal until even middle age. The age of onset may vary both between and within subtypes and even between patients with the same mutation.

By definition, LGMD have in common a predilection for involvement of the proximal musculature in the shoulder and pelvic girdles, but these may be differentially affected, particularly in the early stages, and involvement of distal muscles may also occur. Rate of progression of the muscle weakness may not be linear. Features such as spinal rigidity, scoliosis and limb contractures should be sought. Hypertrophy, usually of calf muscles but also of other limb muscles and even the tongue, may be present. Family history may suggest an autosomal dominant inheritance or consanguinity.

Although it is not possible to provide an absolute prediction of the clinical pattern, Table 2 in the original guideline document outlines the presence or absence of typical features in each LGMD to give a guide to the underlying diagnosis. Exceptions to the commonly recognized patterns can occur and the table should be seen as a guide only. It is also important to point out that for mutations in some of these genes, there is clinical heterogeneity. Specific examples of this include myotilin mutations (responsible for the rare LGMD1A and myofibrillar myopathy), caveolin-3 mutations (reported with a range of presentations including hyperCKaemia, LGMD1C and rippling muscle disease) and lamin A/C mutations, which are probably the most clinically variable of all, and have been reported in at least seven distinct diseases, in some of which muscle involvement may be minimal or absent. The variability in presentation for all of these conditions means that different family members, or indeed the same individual, may present with one or more manifestations of mutation in a particular gene.

Refer to the original guideline document for information on specific clinical pointers/indicators of LGMD.

Investigation

Serum creatine kinase (CK) is a simple and useful investigation provided that non-muscle conditions are excluded first. The degree of elevation may be helpful in differentiating broadly between diagnoses; typically, it may be normal or only mildly raised in conditions such as LGMD1A and 1B, moderately raised (5–10x

upper limit of normal) in LGMD1C, 2A, 2C-F and 2I and grossly raised (>10x) in LGMD2B.

Neurophysiology studies are of little value in refining a diagnosis of LGMD. Nerve conduction studies can exclude a neuropathy if this causes diagnostic doubt in the early stages of presentation. Electromyography usually shows myopathic features in patients with any type of LGMD with no ability to further specify the diagnosis. Laminopathy patients may additionally or exclusively have a peripheral neuropathy.

Muscle imaging with computed tomography or magnetic resonance imaging is used increasingly to determine patterns of muscle involvement. No large studies of the LGMD have been published but case reports and small series suggest characteristic patterns in some conditions. The most consistent examples are LGMD2A which selectively involves hip extensors and adductors, involvement of the glutei in alpha-sarcoglycanopathy and LGMD2J where loss of the thigh muscles and involvement of tibialis anterior is present.

Muscle biopsy site(s) may be guided using imaging results. They will probably yield the most useful information if they are undertaken on a clinically affected muscle but preferably not one that is 'end-stage'. No studies compare open versus needle biopsies, although with the increasing number of immunohistochemical and immunoblotting procedures were possible, it is important to obtain sufficient tissue to allow meaningful interpretation.

Muscle tissue should be analysed firstly with standard histological techniques. All LGMD show dystrophic features with variation in fibre size, increased numbers of central nuclei and endomysial fibrosis. Inflammatory infiltrates are seen most commonly in dysferlin deficiency. Thus there is the potential for diagnostic confusion and patients may have received a previous diagnosis of polymyositis. Rimmed vacuoles and Z-line streaming may be seen in myotilin deficiency. Table 3 of the original guideline document summarizes typical findings in each condition.

Immunohistochemistry and immunoblotting should be undertaken in a laboratory with sufficient expertise in both the performance and interpretation of these techniques. Immunohistochemical staining with a panel of antibodies ideally including all four anti-sarcoglycan antibodies may show one or more abnormalities. Demonstration of normal dystrophin staining is important (although there may be a mild secondary reduction in sarcoglycan deficiency). Quantitative analysis of proteins by Western blotting may be an additional useful technique for elucidating primary and secondary protein abnormalities.

Refer to Table 3 in the original guideline document for a summary of commonly observed primary and secondary changes on immunoanalysis.

Immunoblotting has been the accepted test required for the diagnosis of LGMD2A. However, there is variability in the quantity and function of calpain-3 protein detected on immunoblots, even for those patients in whom a calpain mutation is proven; thus, emphasis may shift to earlier analysis of the calpain-3 gene.

One group has developed a blood-based assay for dysferlin expression in monocytes, showing that this correlates with skeletal muscle expression. This

potentially avoids the need for muscle biopsy although is not in mainstream use at present.

Deoxyribonucleic acid (DNA) analysis directed to provide confirmation of mutation in the affected gene(s) is the gold standard of diagnosis and necessary to be able to offer carrier or pre-symptomatic testing to other family members. This is more straightforward in some forms of LGMD than others, depending to a large extent on whether or not there are commonly detected mutations or if mutations in different families tend to be unique. For example, the FKRP 'common mutation' C826A in LGMD2I can be detected readily in a diagnostic laboratory whereas some of the other causative genes are large (e.g., dysferlin [55 exons]), and screening for mutations is a formidable task. Thus mutation analysis in these genes is, at present, available only in selected laboratories. Mutation detection for the rarer types of LGMD may only be available on a research basis.

Good practice points. Careful clinical assessment of factors such as the pattern of muscle involvement, associated features and family history should suggest probable diagnosis in a patient with LGMD. Confirmation of this should be achieved through the selective use of predominantly laboratory-based investigations, some of which are highly specialised and should only be undertaken in a laboratory with appropriate expertise. In some conditions this may be relatively straightforward but in others verification of the underlying mutation presently remains in the realm of the research laboratory. In the UK, patients may be referred for assessment to the centre for limb girdle muscular dystrophy (n.scag@ncl.ac.uk) funded by the National Specialist Commissioning Advisory Group.

Assessment and Monitoring of Adjunctive Aspects

Respiratory Management

Respiratory muscle weakness resulting in symptomatic hypoventilation and respiratory failure is found in a few of the LGMD, most frequently in LGMD2I and the sarcoglycanopathies. In LGMD2I and occasionally in the sarcoglycanopathies, respiratory failure may arise whilst the patient is still ambulant.

There are no recommendations specific to the LGMD but extrapolation from the monitoring and investigation of respiratory involvement in other neuromuscular conditions is helpful. Awareness of symptoms of respiratory insufficiency such as frequent chest infections, morning headache and daytime somnolence is important. Measurements of sitting and supine if <80% forced vital capacity (FVC) may be made in the outpatient clinic. Overnight pulse oximetry is recommended if the FVC is <60%. Annual influenza vaccination and prompt treatment of respiratory infections are suggested. Liaison with a respiratory physician with experience in the management of neuromuscular disorders is essential to ensure optimal timing of intervention with nocturnal home ventilation.

Cardiac Management

The important issue of cardiac complications in LGMD as well as in other muscle conditions was considered at the 107th European Neuromuscular Centre (ENMC) Workshop. Cardiac involvement may manifest as a conduction defect and/or

cardiomyopathy. In laminopathies, arrhythmias such as atrioventricular block, atrial paralysis and atrial fibrillation/flutter occur in the majority of patients by age 30 years, and permanent pacing is required. However, even with permanent pacing, a recent paper cites a sudden death rate of 46% in lamin A/C mutation carriers and therefore recommends an implantable defibrillator. Dilated cardiomyopathy arises in a third of laminopathy patients and is usually severe. Arrhythmias and hypertrophic or dilated cardiomyopathy are present in approximately 20% of sarcoglycanopathy patients. One-third of LGMD 2I patients have a cardiomyopathy which is symptomatic. The remaining LGMD do not characteristically show significant cardiac compromise.

Thus the ability to define precisely the underlying genetic defect allows a tailored approach to monitoring through better anticipation of the onset and progression of cardiac aspects. Monitoring and treatment of LGMD1B, 2C-F and 2I patients require close cardiological supervision. Electrocardiogram (ECG) and echocardiography are suggested as the standard initial investigations. In the absence of dedicated studies, treatment of heart failure is undertaken on general principles with early use of angiotensin-converting enzyme inhibitors. Anticoagulation may need consideration in patients with atrial fibrillation or standstill. For patients with particularly severe cardiac failure but relatively well-preserved respiratory function, consideration of cardiac transplantation may be appropriate.

Good practice points. Although serial monitoring of basic measurements of respiratory and cardiac function is attainable in the neurology outpatient setting, patients with a LGMD subtype known to place them at additional risk of cardiorespiratory complications ideally should be managed in conjunction with a respiratory physician and/or cardiologist. Intervention in the form of nocturnal ventilatory assistance for respiratory failure and with permanent pacing and/or management of cardiomyopathy may be life saving. The need to monitor for and treat complications as appropriate also applies to those patients in whom the underlying diagnosis is unknown as it follows that the attendant risk of cardiorespiratory complications is also unknown, but that general principles of management will apply.

Physical Management

There are no papers relating specifically to LGMD and physiotherapy, exercise or orthotic use. The application of general principles is probably appropriate. Prevention of contracture development through stretching and splinting orthoses is important in maximizing functional ability. Release of functionally limiting contractures (especially of the Achilles tendons) may be necessary especially in LGMD1B, LGMD2A, in childhood onset sarcoglycanopathy or LGMD2I. Scoliosis in LGMD occurs mainly after wheelchair dependence and attention should be paid to seating. The role of exercise is controversial but basic guidelines as for other types of muscular dystrophy would encourage gentle exercise within comfortable limits and the avoidance of prolonged immobility.

Genetic Counseling

Many patients seek medical advice because of concern for themselves, relatives or descendants. Delineation of the LGMD subtype allows knowledge of its autosomal

dominant or recessive inheritance pattern to inform genetic counselling appropriately. Confirmation of the diagnosis in LGMD2I patients in particular has led to altered advice in some, as previously they had been thought to be affected by Becker muscular dystrophy, an X-linked condition.

Drug Treatment

There are no established drug treatments for the LGMD. Six patients with sarcoglycan-deficient muscular dystrophy took part in a double-blind, placebo-controlled crossover trial of creatine monohydrate. Thirty patients with other conditions were included. The mean improvement of 3% in muscle strength over the 8-week trial period was found to be significant but modest. There are no relevant studies on the use of co-enzyme Q10 (ubiquinone).

Corticosteroids have an established role in Duchenne muscular dystrophy (DMD) boys; on this basis, they have been used empirically in some patients with LGMD2C-F with reported improvement. As these conditions are so much rarer than DMD, it will not be possible to perform adequate treatment trials without collaboration amongst multiple neuromuscular centres. Anti-inflammatory drugs have been suggested to suppress the inflammation seen in LGMD2B muscles. Trials in the animal model of LGMD2B are proposed, and there is a randomized clinical trial underway in Germany.

Definitions:

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Good Practice Points Where there was a lack of evidence but consensus was clear, the Task Force has stated their opinion as good practice points.

CLINICAL ALGORITHM(S)

None provided

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of supporting evidence is identified and graded for selected recommendations (see "Major Recommendations" field).

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Prognosis for limb girdle muscular dystrophies (LGMD) is not uniform; thus, timely intervention through early identification of potential complications may improve survival.

POTENTIAL HARMS

Not stated

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

The European Federation of Neurological Societies has a mailing list, and all guideline papers go to national societies, national ministries of health, World Health Organisation, European Union, and a number of other destinations. Corporate support is recruited to buy large numbers of reprints of the guideline papers, and permission is given to sponsoring companies to distribute the guideline papers from their commercial channels, provided there is no advertising attached.

IMPLEMENTATION TOOLS

Staff Training/Competency Material

For information about [availability](#), see the "Availability of Companion Documents" and "Patient Resources" fields below.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Living with Illness

IOM DOMAIN

Effectiveness
Patient-centeredness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

Norwood F, de Visser M, Eymard B, Lochmuller H, Bushby K, EFNS Guideline Task Force. EFNS guideline on diagnosis and management of limb girdle muscular dystrophies. *Eur J Neurol* 2007 Dec;14(12):1305-12. [28 references] [PubMed](#)

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2007 Dec

GUIDELINE DEVELOPER(S)

European Federation of Neurological Societies - Medical Specialty Society

SOURCE(S) OF FUNDING

European Federation of Neurological Societies

GUIDELINE COMMITTEE

European Federation of Neurological Societies Task Force on the Management of Limb Girdle Muscular Dystrophies

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

Task Force Members: F. Norwood, Institute of Human Genetics, Newcastle upon Tyne, UK; King's Neuroscience Centre, King's College Hospital, London, UK; M. de Visser, Department of Neurology, Academic Medical Centre, University of Amsterdam, Amsterdam, Holland; B. Eymard, Hôpital de la Pitié, Salpêtrière, Paris, France; H. Lochmüller, Institute of Human Genetics, Newcastle upon Tyne, UK; Genzentrum, Ludwig-Maximilians-Universität, Munich, Germany; K. Bushby, Institute of Human Genetics, Newcastle upon Tyne, UK

FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

None declared

GUIDELINE STATUS

This is the current release of the guideline.

Because of the rate of advance in this group of conditions, it is suggested that these guidelines are reviewed after 2 years.

GUIDELINE AVAILABILITY

Electronic copies: Available to registered users of the [European Federation of Neurological Societies Web site](#).

Print copies: Available from Prof. K. Bushby, Institute of Human Genetics, Central Parkway, Newcastle upon Tyne, NE1 3BZ, UK; Phone: +44191 241 8757; Fax: +44191 241 8799; E-mail: kate.bushby@ncl.ac.uk.

AVAILABILITY OF COMPANION DOCUMENTS

The following are available:

- Brainin M, Barnes M, Baron JC, Gilhus NE, Hughes R, Selmaj K, Waldemar G; Guideline Standards Subcommittee of the EFNS Scientific Committee. Guidance for the preparation of neurological management guidelines by EFNS scientific task forces – revised recommendations 2004. Eur J Neurol. 2004 Sep;11(9):577-81. Electronic copies: Available in Portable Document Format (PDF) from the [European Federation of Neurological Societies Web site](#).
- Guideline papers. European Federation of Neurological Societies. Electronic copies: Available from the [European Federation of Neurological Societies Web site](#).
- Continuing Medical Education questions available from the [European Journal of Neurology Web site](#).

PATIENT RESOURCES

None available

NGC STATUS

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