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Teaching Course 10

Clinical science in muscle disorders (Level 2)

Differential diagnosis of "scapular winging"

Antonio Toscano
Messina, Italy

Email: atoscano@unime.it

Differential diagnosis of "Scapular Winging"

A. Toscano

Head of European Reference Center (ERN EURO-NMD) for Neuromuscular Disorders,
UOC Neurologia e Malattie Neuromuscolari
University of Messina, Italy

DISCLOSURES

Prof. A. Toscano has received reimbursement for educational motivations and having served as a member of scientific boards for Sanofi Genzyme, Amicus, Spark and CSL Behring

Scapular dyskinesis

Collective term that refers to movements of a “dysfunctional scapula”

Scapular dyskinesis has been defined as:

1) abnormal static scapular position and/or dynamic scapular motion, characterized by medial/lateral border prominence; 2) Inferior angle prominence and/or early scapular elevation; 3) rapid downward rotation during arm lowering.

Scapular dyskinesis can also be a painful condition

There are multiple causative factors, both proximally (muscle weakness, nerve injury) and distally (acromioclavicular joint injury, superior labral tears, rotator cuff injury).

Dyskinesis can alter the roles of the scapula in the “scapulo–humeral rhythm”: dynamic interaction between the scapula and the humerus.

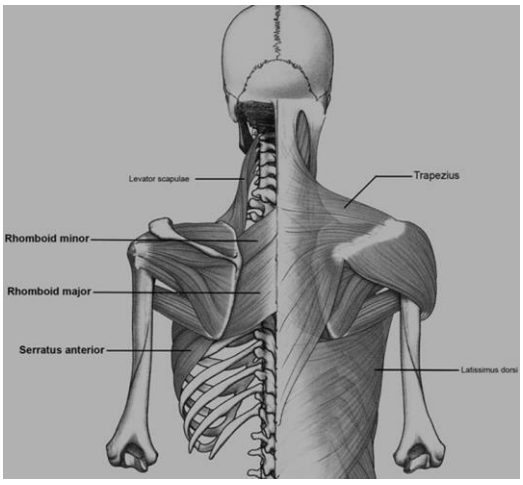
This can be due to alterations in the bony stabilizers, alterations in muscle activation patterns or strength in the dynamic muscle stabilizers.

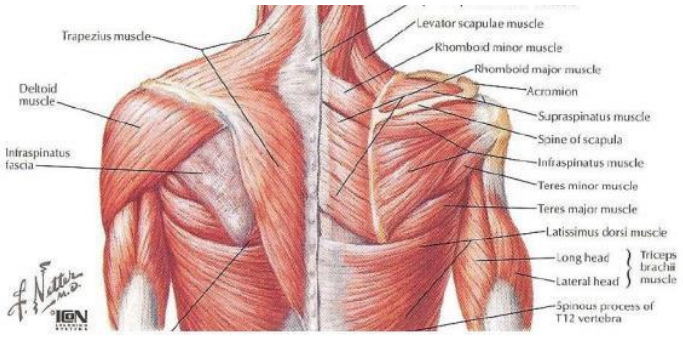
Scapular Winging: a dysfunction involving the stabilizing muscles of the scapula resulting in inbalance and abnormal motion of the scapula

Why the patients refer to a physician because of a Scapular Winging ?

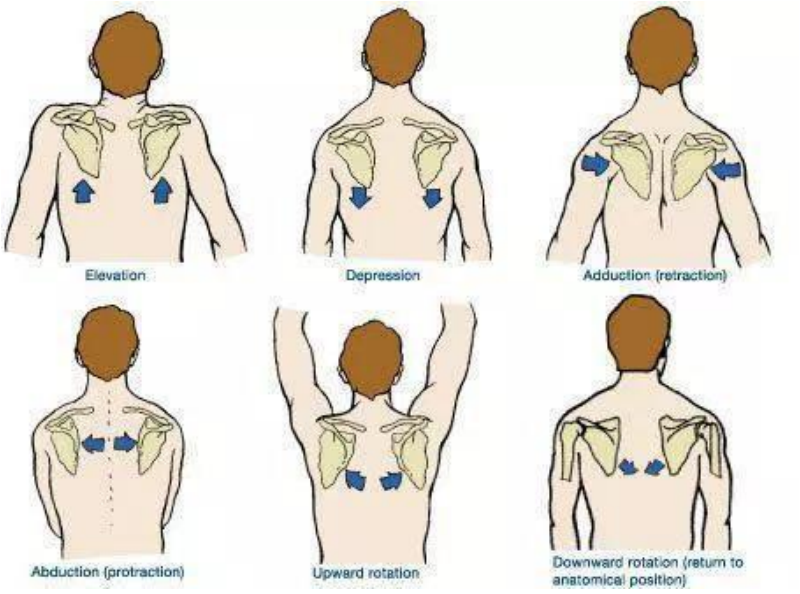
- Muscle weakness
- Pain
- Aesthetic change

Scapular anatomy and its relationships





Scapular movements



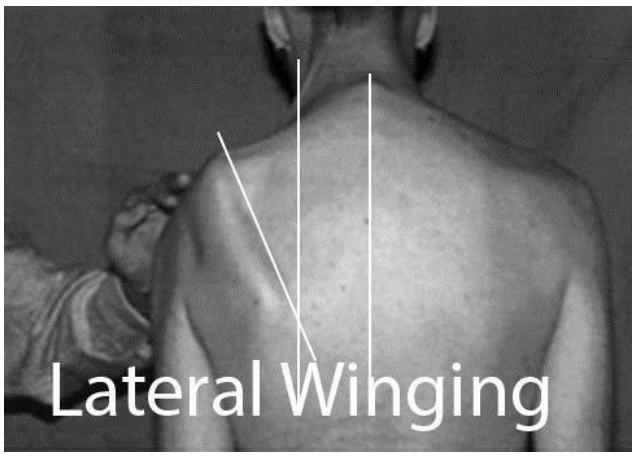
Main causes of Scapular Winging

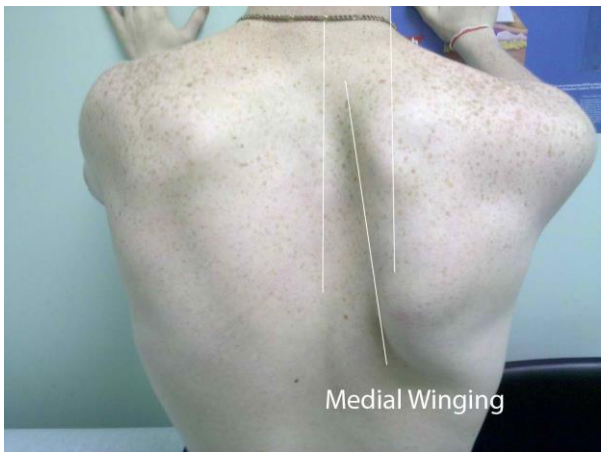
Traumatic

- Nerves injuries
- Muscles trauma
- Attachments alterations

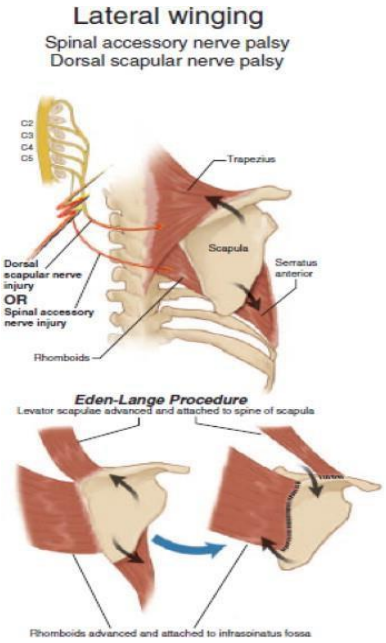
Non Traumatic

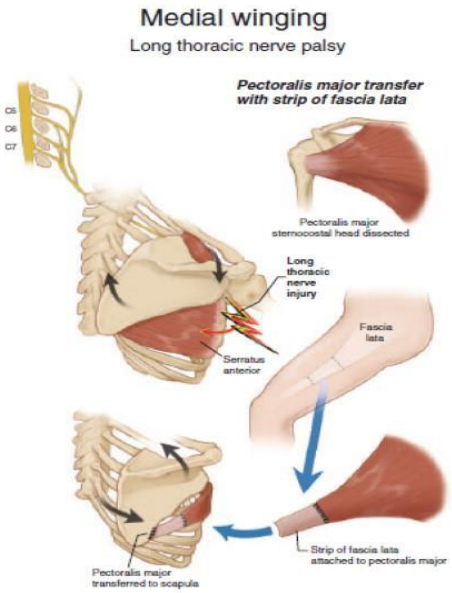
- Structural anomalies of the scapula
- Osteochondroma
- Fractures
- Neurogenic causes
- Muscle disorders





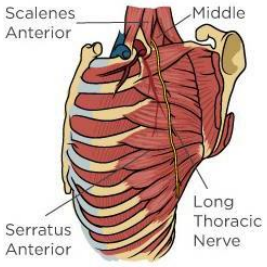
Scapular movements abnormalities

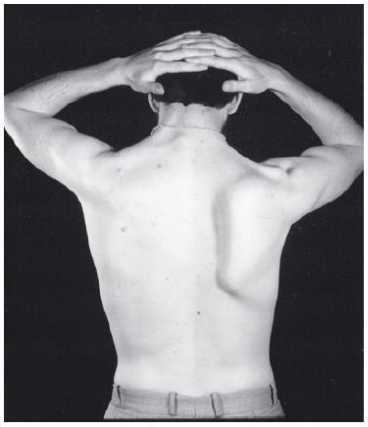




Neurogenic causes of scapular winging

	Medial winging	Lateral winging	
Injured nerve	Long thoracic	Spinal accessory	Dorsal scapular
Muscle palsy	Serratus anterior	Trapezius	Rhomboids
Physical exam	Arm flexion; push-up motion against a wall	Arm abduction; external rotation against resistance	Arm extension from full flexion
Position of the scapula compared to normal	Entire scapula displaced more medial and superior	Superior angle more laterally displaced	Inferior angle more laterally displaced





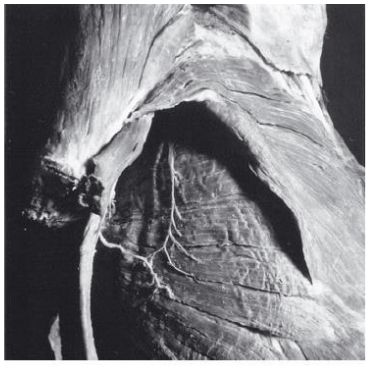


Fig. 2 The long thoracic nerve with branches to each digitation of the serratus anterior.

The Long Thoracic Nerve originates from branches of the C5 and C6 nerve roots. These branches join beneath the middle scalene muscle and with some variability, either pierce the middle scalene or emerge between the middle and anterior scalene before uniting with a branch of the C7 (and sometimes C8) nerve root. The long thoracic nerve travels through the axilla to innervate the serratus anterior muscle- a shoulder protractor and scapular stabilizer. Injury to the long thoracic nerve denervates the serratus anterior muscle, resulting in scapular winging and shoulder instability.

Case report

R.G., 38 yrs, M

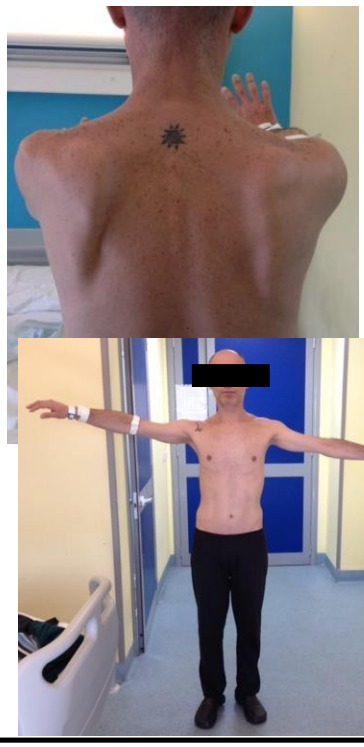
Diabetes Mellitus type 1
Basedow-Graves disease
Crohn's disease

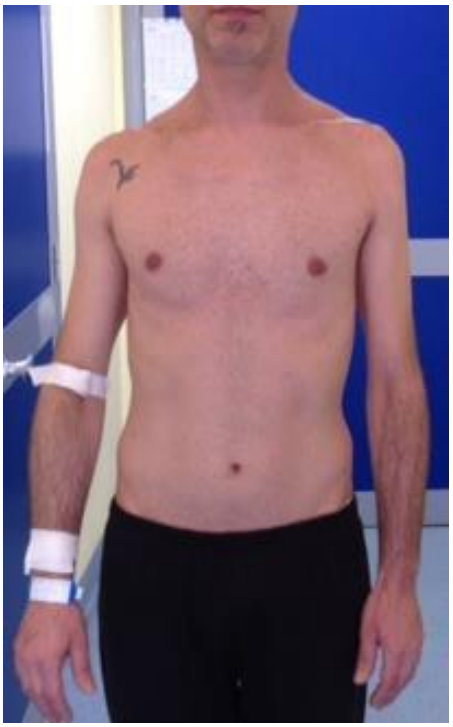
Four months before the admission to the hospital, sudden onset of shoulder and left arm intense pain. Later on, progressive proximal muscle weakness and wasting with paresthesias

Neurological examination at admission

- Wasting of supraspinatus, deltoid, dentate, brachial biceps, triceps, flexors and extensors antibrachiali on the left
- Frequent fasciculations of the affected muscles
- Intense myalgia at rest and/or after abduction of left upper limb
- OT reflexes: normal

Gruppo muscolare	Dx	Sn
Flessori del collo	5	5
Estensori del collo	5	5
Abduttori della spalla	5	3
Rotatori della spalla (laterali esterni)	5	3
Adduttori orizzontali della spalla	5	3
Flessori del gomito	5	4-
Estensori del gomito	5	4-
Estensori del polso	5	4
Flessori del polso	5	4
Estensore comune delle dita	5	4+
Flessori delle dita	5	5
Estensori dell'anca	5	5
Adduttori dell'anca	5	5
Abduttori dell'anca	5	5
Flessori dell'anca	5	5
Estensori del ginocchio	5	5
Flessori del ginocchio	5	5
Flessori dorsali del piede	5	5
Eversori del piede	5	5
Inversori del piede	5	5
Flessori plantari del piede	5	5
Flessore dorsale I dito	5	5
Flessori dorsali dita	5	5
Flessori plantari dita	5	5
Orbicolari palpebre	5	5
Peribuccali	5	5





Laboratory examination

Blood routine analyses: normal apart from HbA1C (8.5%)

Brain and cervical spine MRI: normal

EMG/ENG: neurogenic pattern with denervation and collateral reinnervation on left supraspinatus, deltoid, tricipes and brachial biceps muscles.

MEPs: normal

Cerebrospinal fluid examination: 1 cell (n.v 0-5), 80 mg/dl proteins (n.v 0-45)

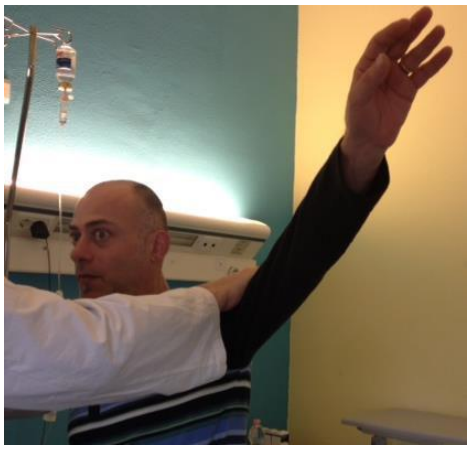
DIAGNOSIS: Disimmune plexopathy (Parsonage - Turner syndrome)

THERAPY: IVIG (0,4 g/Kg/die for 5 days)

Gruppo muscolare	Dx	Sn
Flessori del collo	5	5
Estensori del collo	5	5
Abduttori della spalla	5	4+
Rotatori della spalla (laterali esterni)	5	4+
Rotatori interni della spalla	5	4 +
Adduttori orizzontali della spalla	5	4+
Flessori del gomito	5	4+
Estensori del gomito	5	4+
Estensori del polso	5	4
Flessori del polso	5	4
Estensore comune delle dita	5	4+
Flessori delle dita	5	5
Estensori dell'anca	5	5
Adduttori dell'anca	5	5
Abduttori dell'anca	5	5
Flessori dell'anca	5	5
Estensori del ginocchio	5	5
Flessori del ginocchio	5	5
Flessori dorsali del piede	5	5
Eversori del piede	5	5
Inversori del piede	5	5
Flessori plantari del piede	5	5
Flessore dorsale I dito	5	5
Flessori dorsali dita	5	5
Flessori plantari dita	5	5
Orbicolari palpebre	5	5
Peribuccali	5	5

Neurological examination after IVIG couses

- Myalgia markedly reduced during left arm abduction
- Strenght of left arm abductors and brachial biceps very much increased



Parsonage-Turner syndrome

Parsonage-Turner syndrome (PTS) is characterized by a rapid onset of severe pain in the shoulder and arm.

The acute period may last for few hours till few weeks.

Usually, patients may experience wasting and weakness (Neuralgic Amyotrophy) of the affected muscles.

PTS involves mainly the brachial plexus.

The involved nerves control movements and sensations in the shoulders, arms, elbows, hands, and wrists.

Exact causes of PTS are unknown, but it could be related to alterations of the immune system (immune-mediated disorder).

Affected individuals may recover after specific treatment: strength returns to the affected muscles and pain goes away. Sometimes, a positive evolution occurs without treatment

However, some affected individuals may experience residual pain and potentially significant disability.

Case report

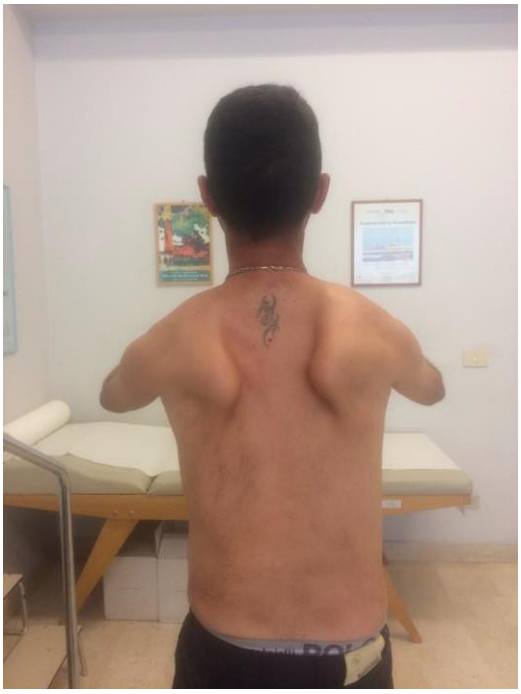
Male, 47 years of age,

No family history of neuromuscular disorders. Parents not consanguineous.

At the age of 39 years, fatigability in running and in climbing the stairs, muscle cramps, fasciculations, and scapular winging.

In the following years, increased muscle involvement, predominantly affecting upper limbs, pelvic girdle and legs antero-lateral compartment muscles. At 44 years of age, difficulties in swallowing.

At admission, neurological examination showed: waddling gait with bilateral “steppage”, not able to raise from the floor and needed support to stand up from a chair and from the bed



Neurological examination: Scapular Winging and severe muscle atrophy in the legs (predominantly at the anterior compartment). Marked weakness of the periscapular muscles with severe limitation of the arms abduction bilaterally; weakness (MRC 3) also present at the following muscles: iliopsoas, gluteus, tibialis anterior bilaterally. OT reflexes: brisk at upper limbs, absent at lower limbs.

Laboratory investigations

CK: 204 U/l

EMG: a neurogenic pattern with hallmarks of denervation and collateral re-innervation in all examined muscles

MEPs: normal

Muscle biopsy (vastus lateralis muscle): variability of fiber size, muscle grouping and several angulated fibers

NGS: a homozygous mutation in the valosin-containing protein (*VCP*) gene (NM-00716:C.463T:p.R55C).

Diagnosis: Lower Motor Neuron disease VCP-related

This mutation has been associated with atypical forms of amyotrophic lateral sclerosis with variable degree of cognitive impairment and in inclusion body myopathy with Paget disease of the bone and frontotemporal dementia (IBMPFD).

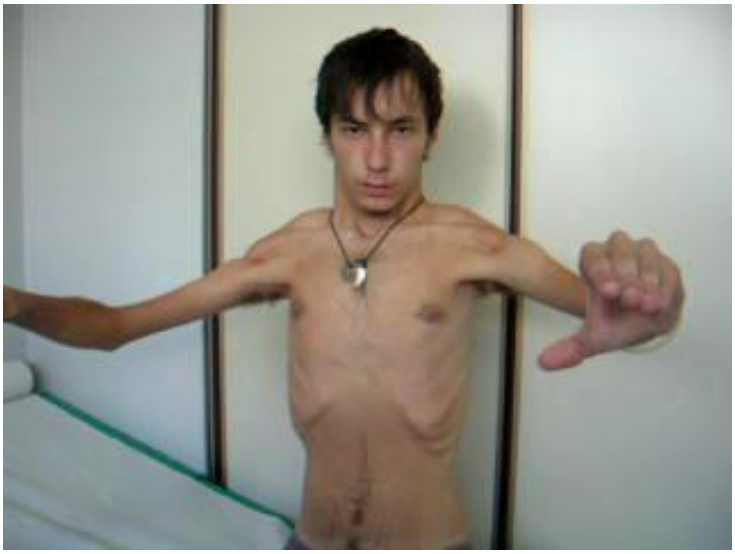
Muscle disorders

- Dystrophy
- FSH: Often asymmetric
- LGMD
  - 1F: TNPO3
  - 2A: Calpain 3
  - 2D: α-Sarcoglycan
  - 2E: β-Sarcoglycan
  - 2I: FKRP
  - 2N: POMT2
  - 2S: TRAPPC11
  - 2Z: POGLUT1
- Emery-Dreifuss
- Myopathy + Paget's disease of bone
  - with Dementia (VCP)
  - Type 2
- Desmin myopathy
  - Early onset,Recessive
  - Kaeser, Dominant

Scapuloperoneal syndromes

ACTA1: 1q42;
Centronuclear myopathy (Adult-onset): MYF6; 12q21;
Davidenkow's syndrome
Emery-Dreifuss Dystrophies
FSHD with ragged red fibers & cardiomyopathy
Glycogen storage
Acid maltase deficiency with scapuloperoneal weakness
Phosphorylase deficiency (McArdles)
Kaeser: Desmin; 2q35
Myopathy + Paget's disease with Dementia: VCP; 9p13
Retardation & Cardiomyopathy: LAMP-2; Xq24
Scapuloperoneal MD (SPMD) with Hyaline bodies
Type 1: FHL1; Xq26; Type 2: MYH7; 14q12; Type 3: MYH7; 14q12;
Myosin storage myopathy: MYH7; 14q12
Scapuloperoneal neuronopathy: TRPV4; 12q24

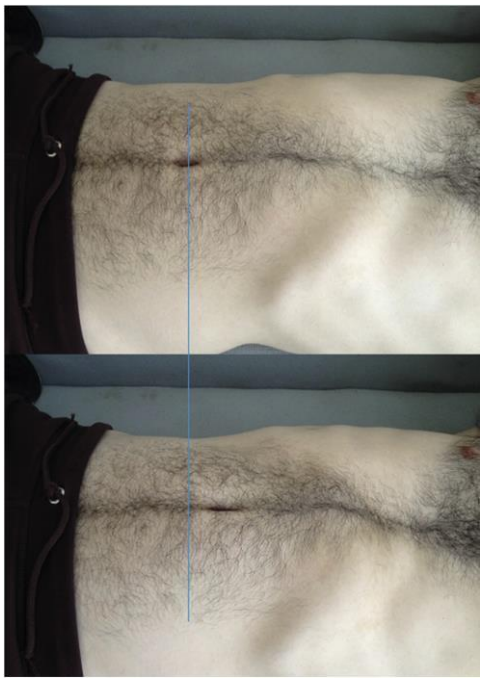
Others neuromuscular forms



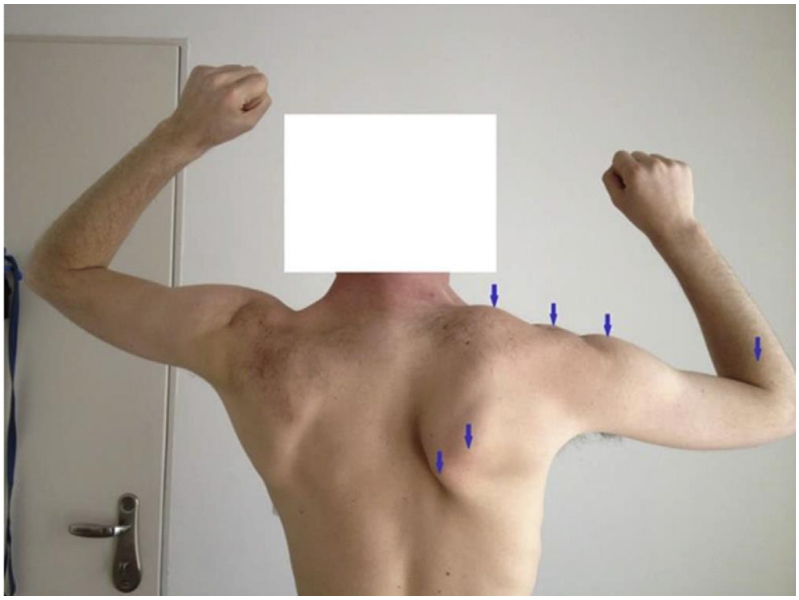
FSHD

- Third most common muscular dystrophy
  - prevalence of ~1:15,000-20,000
- Age of onset is variable from presentations at birth to late life
- Penetrance is high with 95% of patients manifesting weakness by age 20
- Classically, the disease presents with facial, proximal arm weakness with winged scapula followed by weakness of foot dorsiflexion and hip girdle muscles
- Asymmetric involvement is frequent
- Bulbar, extraocular, and respiratory muscles tend to be spared
- 20% of the patients wheelchair bound and 1% with respiratory weakness

- Typical symptoms (facial or scapulo-humeral weakness) on presentation in ~70-85%
- No facial involvement in 6-18%
- Case reports of:
  - Facial-sparing scapular myopathy
  - Limb-girdle weakness
  - Late-onset distal myopathy after age 50
  - Asymmetric brachial weakness
  - Isolated axial weakness
  - Monomelic lower limb atrophy



Beevor's sign



Poly-Hill Sign

J Neurol (2016) 263:1204–1214
DOI 10.1007/s00415-016-8123-2

ORIGINAL COMMUNICATION

A novel clinical tool to classify facioscapulohumeral muscular dystrophy phenotypes

Giulia Ricci[1,2] · Lucia Ruggiero[3] · Liliana Vercelli[4] · Francesco Sera[5] · Ana Nikolic[1] · Monica Govi[1] · Fabiano Mele[1] · Jessica Daolio[1] · Corrado Angelini[6] · Giovanni Antonini[7] · Angela Berardinelli[8] · Elisabetta Bucci[7] · Michelangelo Cao[9] · Maria Chiara D'Amico[10] · Grazia D'Angelo[11] · Antonio Di Muzio[10] · Massimiliano Filosto[12] · Lorenzo Maggi[13] · Maurizio Moggio[14] · Tiziana Mongini[4] · Lucia Morandi[13] · Elena Pegoraro[9] · Carmelo Rodolico[15] · Lucio Santoro[3] · Gabriele Siciliano[2] · Giuliano Tomelleri[16] · Luisa Villa[14] · Rossella Tupler[1,17]

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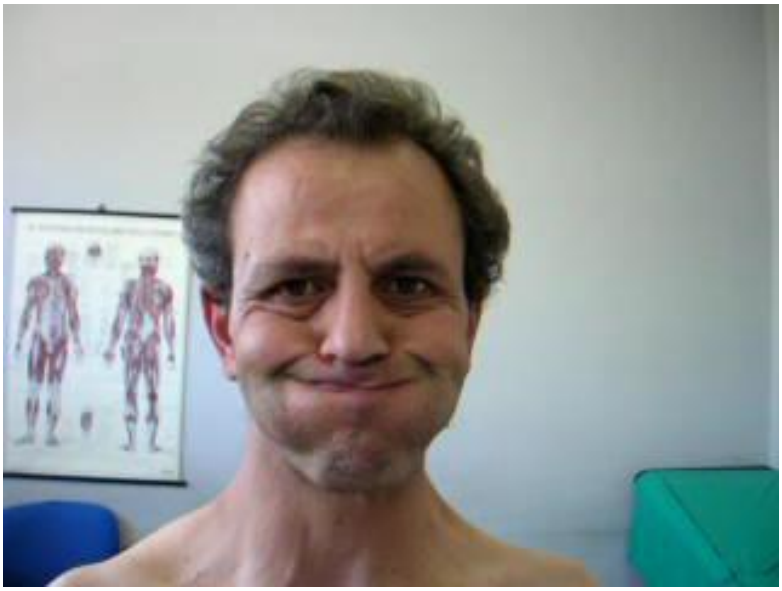
Abstract Based on the 7-year experience of the Italian Clinical Network for FSHD, we revised the FSHD clinical form to describe, in a harmonized manner, the phenotypic spectrum observed in FSHD. The new Comprehensive Clinical Evaluation Form (CCEF) defines various clinical categories by the combination of different features. The inter-rater reproducibility of the CCEF was assessed between two examiners using kappa statistics by evaluating 56 subjects carrying the molecular marker used for FSHD diagnosis. The CCEF classifies: (1) subjects presenting facial and scapular girdle muscle weakness typical of FSHD (category A, subcategories A1–A3), (2) subjects with muscle weakness limited to scapular girdle or facial muscles (category B subcategories B1, B2), (3) asymptomatic/healthy subjects (category C, subcategories C1, C2), (4) subjects with myopathic phenotype presenting clinical features not consistent with FSHD canonical phenotype (D, subcategories D1, D2). The inter-rater reliability study showed an excellent concordance of the final four CCEF categories with a κ equal to 0.90; 95 % CI (0.71; 0.97). Absolute agreement was observed for cate-

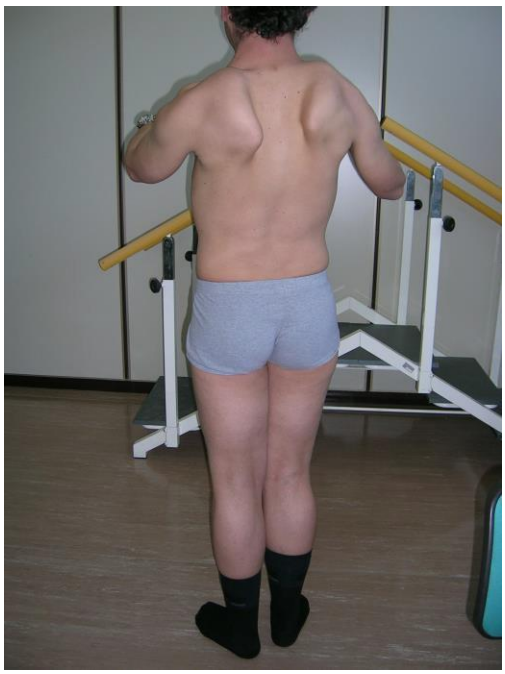
L. Ruggiero and L. Vercelli contributed equally to this work.

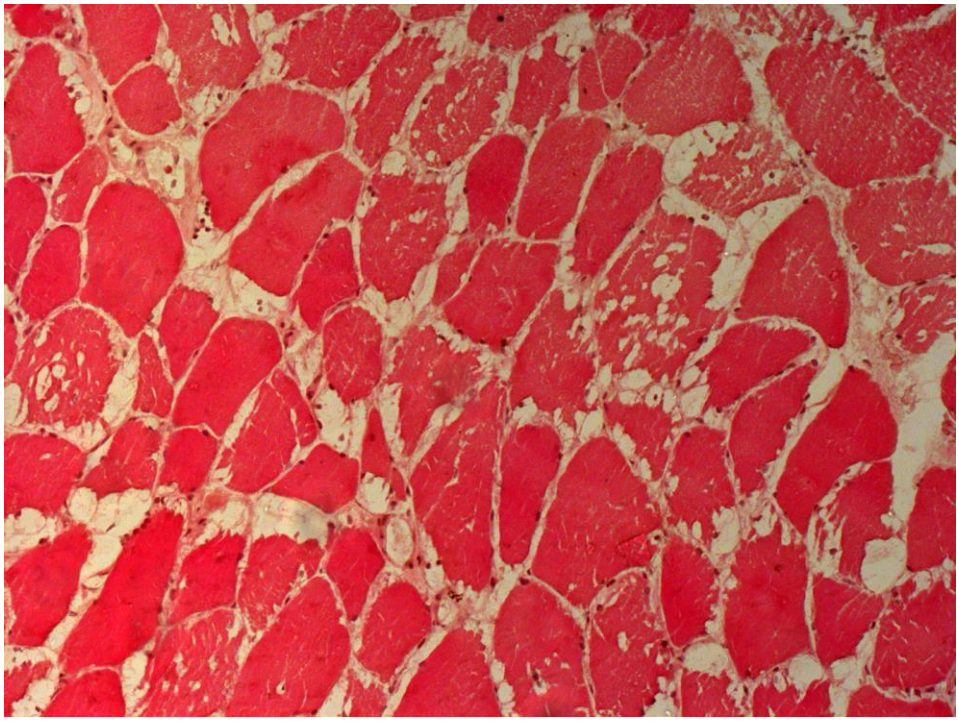
Electronic supplementary material The online version of this article (doi:10.1007/s00415-016-8123-2) contains supplementary material, which is available to authorized users.

G.A., 40 year-old, male

- Parents not consanguineous; no family history of neuromuscular disorders
- Since 20 years of age, unilateral (dx) scapular winging and weakness at upper right limb
- Since 32 years of age, bilateral upper limb weakness
- At 36 years of age, mild distal weakness lower limbs
- CK 504 U/l
- EMG: small MUPs .
- EKG: normal







Laboratory examination

Normal IHC for dystrophin, sarcoglycans, dysferlin, caveolin, desmin

WB for calpain and dysferlin normal

Large presence of vacuoles

GAA, glycolytic and glycogenolytic enzyme activities: normal

Genetic analysis: PFGE analysis of DNA fragments digested with EcoRI and EcoRI/Bln1 and hybridated with p13E-11 showed a 35Kb allele of 4qcr compatible with FSHD diagnosis

Diagnosis

Facio-Scapulo-Humeral Muscular Dystrophy with atypical morphological aspects

J Neurol (2010) 257:1108–1118
DOI 10.1007/s00415-010-5471-1

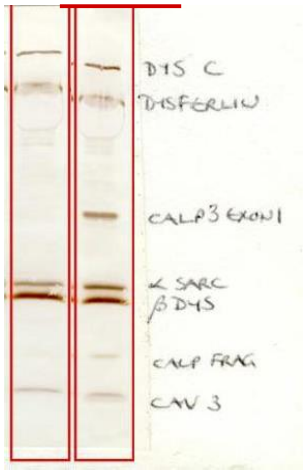
ORIGINAL COMMUNICATION

Facioscapulohumeral muscular dystrophy presenting with unusual phenotypes and atypical morphological features of vacuolar myopathy

Peter Reilich · Nicolai Schramm · Benedikt Schoser · Peter Schneiderat · Nicola Strigl-Pill · Josef Müller-Höcker · Wolfram Kress · Andreas Ferbert · Sabine Rudnik-Schöneborn · Johannes Noth · Hanns Lochmüller · Joachim Weis · Maggie C. Walter

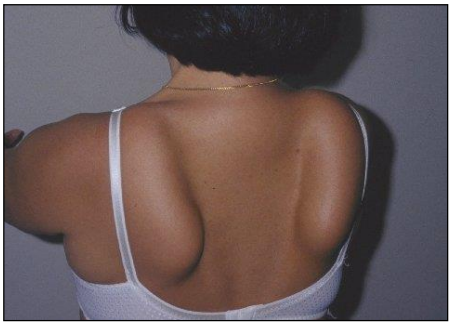
Received: 23 September 2009 / Revised: 15 January 2010 / Accepted: 15 January 2010 / Published online: 10 February 2010

LGMD2A
CAPN3 gene mutation: Homozygous 1981delA



Heart: normal function

FVC: 2.28 litres, 92%

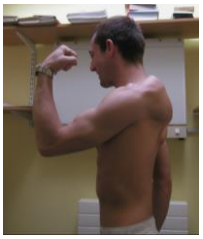


Limb-Girdle Muscular dystrophy due to Calpain deficiency (LGMD2A)
Prevalence: 1-9/100000

Autosomal recessive limb-girdle muscular dystrophy
Variable age of onset

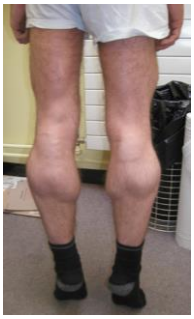
Progressive, typically symmetrical and selective weakness and atrophy of proximal shoulder- and pelvic-girdle muscles (gluteus maximus, thigh adductors, and muscles of the posterior compartment of the limbs mainly affected)
Usually no cardiac or facial involvement.
Main clinical manifestations are exercise intolerance, waddling gait, scapular winging and calf pseudo-hypertrophy.

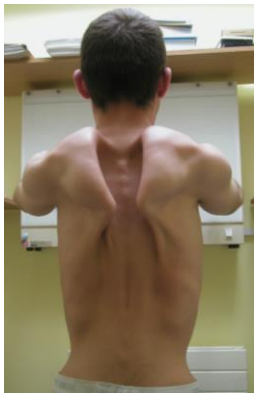
LGMD2D
SGCA gene mutation: 371C>T & 739G>A



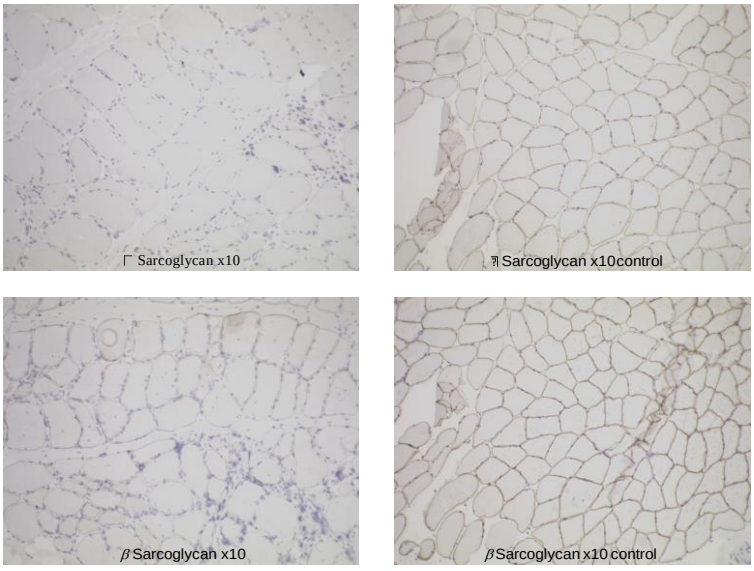
Heart: normal function

FVC: 2.77 litres, 59%





Courtesy
GL Vita
V. Straub



Limb-girdle muscular dystrophy due to alpha-sarcoglycan deficiency (LGMD2D)

Autosomal recessive limb-girdle muscular dystrophy type 2D (LGMD2D)
Prevalence: unknown

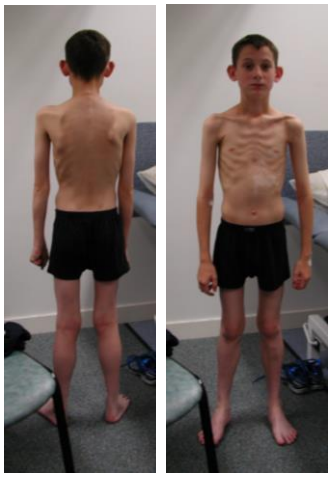
Adolescent or childhood onset

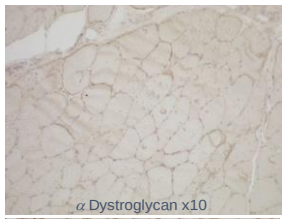
Progressive proximal weakness of the shoulder and pelvic girdle muscles
Main clinical aspects: difficult walking, scapular winging, calf hypertrophy and Achilles tendon contractures, often leading to a tiptoe gait pattern.
Cardiac and respiratory involvement is quite rare

LGMD2I
FKRP gene mutation: 826C>A & 1381G>C

Heart: normal function

FVC: 2.97 litres, 87%





Courtesy
GL Vita
V. Straub

Limb-Girdle Muscular dystrophy due to FKRP deficiency (LGMD2I)

An autosomal recessive limb-girdle muscular dystrophy type 2I

Highly variable age of onset
Slowly progressive proximal weakness of the pelvic and shoulder girdle musculature (predominantly affecting the lower limbs)

Main clinical manifestations: waddling gait, scapular winging, calf and tongue hypertrophy, exercise-induced myalgia, myoglobinuria and/or elevated creatine kinase serum levels.
Abdominal muscle weakness, cardiomyopathy, respiratory muscle involvement and various brain abnormalities reported.

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Neuromuscular Disorders 26 (2016) 312–315

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Case report

TRPV4 related scapuloperoneal spinal muscular atrophy: Report of an Italian family and review of the literature

F. Biasini [a], S. Portaro [b], A. Mazzeo [a], G. Vita [a], G.M. Fabrizi [c,d], F. Taioli [c,d], A. Toscano [a], C. Rodolico [a,*]

[a] Department of Neurosciences, University of Messina, Messina, Italy
[b] IRCCS Centro Neurolesi "Bonino Pulejo", Messina, Italy
[c] Department of Neurological, Biomedical and Movement Sciences, University of Verona, Verona, Italy
[d] Department of Neurosciences, AOUI, Verona, Italy

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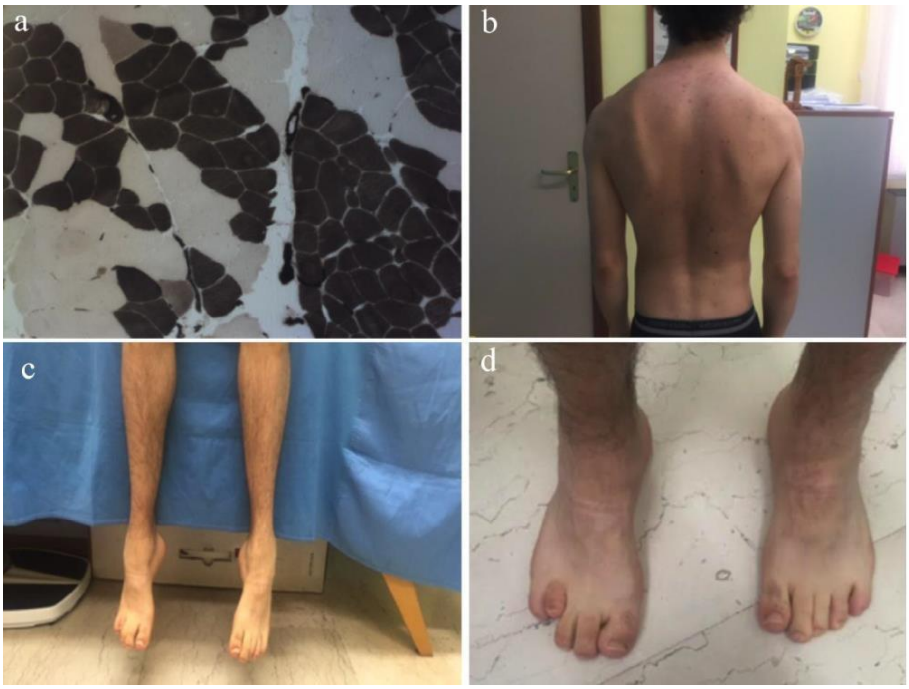
Abstract

Scapuloperoneal spinal muscular atrophy (SPSMA) is a rare autosomal dominant disorder caused by heterozygous mutations in the transient receptor potential cation channel (*TRPV4*) gene, characterized by progressive scapuloperoneal atrophy and weakness. Additional features, such as vocal cord paralysis, scoliosis and/or arthrogryposis, are likely to occur. We report the first Italian family with SPSMA, harboring the c.806G>A mutation in *TRPV4* gene (p. R269H). The pattern of expression was variable: the father showed a mild muscular involvement, while the son presented at birth skeletal dysplasia and a progressive course. We reinforce the concept that the disease can be more severe in the following generations. The disorder should be considered in scapuloperoneal syndromes with autosomal dominant inheritance and a neurogenic pattern. The presence of skeletal deformities strongly supports this suspicion. An early diagnosis of SPSMA may be crucial in order to prevent the more severe congenital form.



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Neuromuscular Disorders 22 (2012) 755–758
www.elsevier.com/locate/nmd

Case report

Winged scapula in patients with myotonic dystrophy type 1 ☆

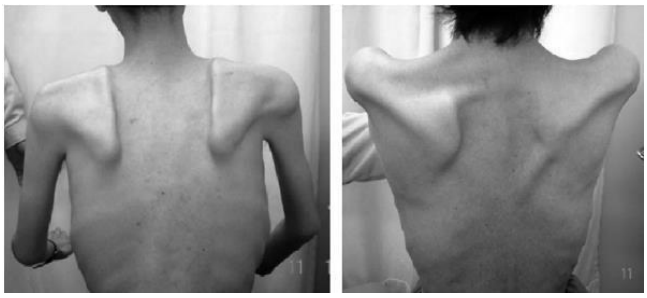
Tadanori Hamano [a,*], Tatsuro Mutoh [a,c], Mikio Hirayama [a,d], Hidemasa Uematsu [b], Itsuro Higuchi [e], Hiroshi Koga [c], Fujio Umehara [e], Kiyonobu Komai [f], Masaru Kuriyama [a]

[a] Second Department of Internal Medicine, Faculty of Medical Sciences, University of Fukui, Fukui, Japan
[b] Department of Radiology, Faculty of Medical Sciences, University of Fukui, Fukui, Japan
[c] Department of Neurology, Fujita Health University, Toyoake, Japan
[d] Department of Neurology, Kanagai Municipal Hospital, Kanagai, Japan
[e] Third Department of Internal Medicine, Kagoshima University School of Medicine, Kagoshima, Japan
[f] Department of Neurology, National Hospital Organization Iou National Hospital, Kanazawa, Japan

Received 24 October 2011; received in revised form 21 February 2012; accepted 27 April 2012

Abstract

We report two patients with myotonic dystrophy type 1 (DM1) showing winged scapula in a single family. Genomic analysis revealed a marked expansion of CTG repeats in the 3′ untranslated region; 1100 in patient 1 and 667 in patient 2. Muscle MRI revealed marked atrophy in the serratus anterior muscle in both patients. Muscle biopsy findings showed central nuclei and variations in fiber size. One of the patients showed ragged red fibers in muscles of the biceps brachii. To our knowledge, this is the first report of typical winged scapula in DM1.



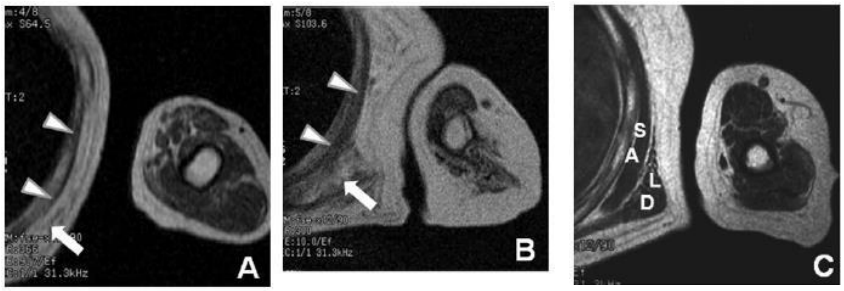


Fig. 2. Muscle MRI findings of the upper thoracic level. Atrophy of the serratus anterior (SA: arrow heads) and latissimus dorsi (LD: arrow) muscles was observed in patient 1 (A) and patient 2 (B). Muscle MRI of the thoracic level in a myotonic dystrophy type1 patient (60-year-old man)

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Case report

50 years to diagnosis: Autosomal dominant tubular aggregate myopathy caused by a novel *STIM1* mutation

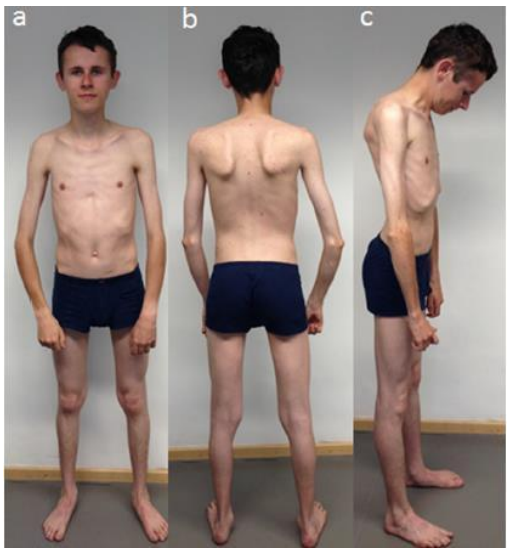
Maggie C. Walter [a,*], Martina Rossius [b], Manuela Zitzelsberger [a], Matthias Vorgerd [c], Wolfgang Müller-Felber [d], Birgit Ertl-Wagner [e], Yaxin Zhang [b], Heinrich Brinkmeier [b], Jan Senderek [a], Benedikt Schoser [a]

[a] Friedrich-Baur-Institute, Department of Neurology, Ludwig-Maximilians-University of Munich, Munich, Germany
[b] Institute of Pathophysiology, Ernst-Moritz-Arndt-University Greifswald, Greifswald, Germany
[c] Department of Neurology, Ruhr-University of Bochum, Bochum, Germany
[d] Department of Neuropaediatrics, Dr.-von-Hauner'sches Kinderspital, University of Munich, Munich, Germany
[e] Department of Radiology, Ludwig-Maximilians-University of Munich, Munich, Germany

Received 4 October 2014; received in revised form 6 April 2015; accepted 8 April 2015

Abstract

Tubular aggregates in human muscle biopsies have been reported to occur in a variety of acquired and hereditary neuromuscular conditions since 1964. Recently mutations in the gene encoding the main calcium sensor in the sarcoplasmic reticulum, stromal interaction molecule 1 (STIM1), have been identified as a cause of autosomal dominant tubular aggregate myopathy. We studied a German family with tubular aggregate myopathy and defined cellular consequences of altered STIM1 function. Both patients in our family had early progressive myopathy with proximal paresis of arm and leg muscles, scapular winging, ventilatory failure, joint contractures and external ophthalmoplegia. One patient had a well-documented disease course over 50 years. Sequencing of the *STIM1* gene revealed a previously unreported missense mutation (c.242G>A; p.Gly81Asp) located in the first calcium binding EF domain. Functional characterization of the new *STIM1* mutation by calcium imaging revealed that calcium influx was significantly increased in primary myoblasts of the index patient compared to controls pointing at a severe alteration of intracellular calcium homeostasis. This new family widens the spectrum of *STIM1*-associated myopathies to a more severe phenotype.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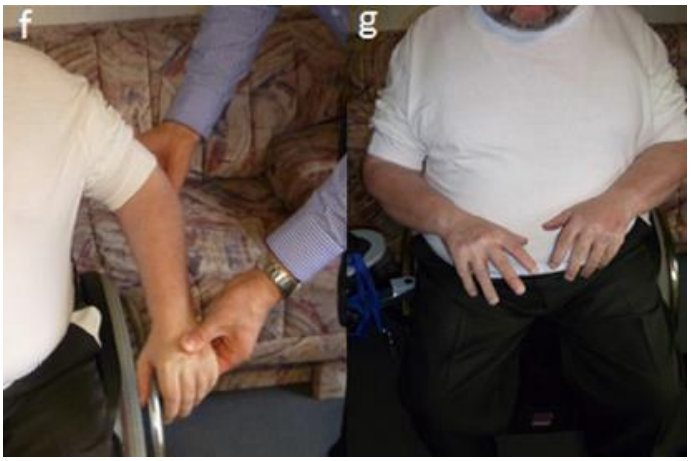
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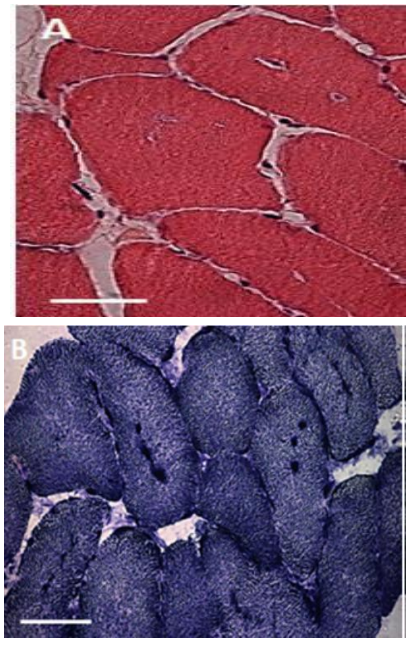
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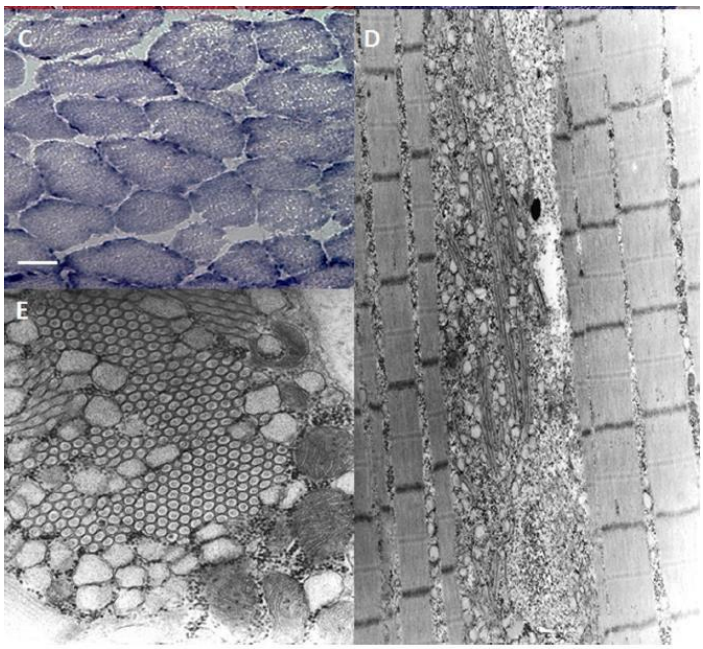
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Osteochondroma as a cause of scapular winging in an adolescent: a case report and review of the literature

Claudio Chillemi[1*], Vincenzo Franceschini[2], Giorgio Ippolito[2], Roberto Pasquali[3], Renato Diotallevi[1], Vincenzo Petrozza[4] and Carlo Della Rocca[4]

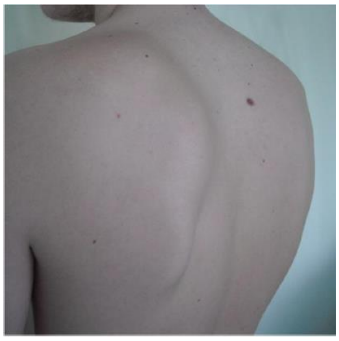
Abstract

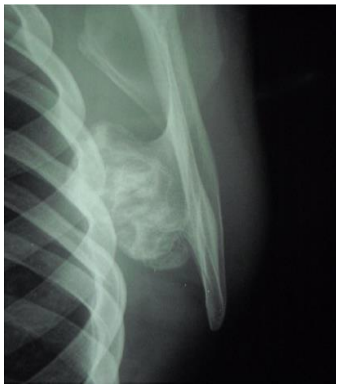
Introduction: Winged scapula is defined as the prominence of the medial border of the scapula. The classic etiopathology of scapular winging are injuries to the spinal accessory or long thoracic nerves resulting respectively in trapezius and serratus anterior palsy. To the best of our knowledge, there are only few reports of scapular lesions being mistaken for winging of the scapula. We report a rare case of a large scapular osteochondroma arising from the medial border and causing a pseudowinging of the scapula.

Case presentation: A 17-year-old Caucasian boy came to us complaining about a winged left scapula. The patient had a complete painless range of motion, but a large hard bony swelling was palpable along the medial border of his left scapula. A grating sensation was felt when his arm was passively abducted and/or elevated causing discomfort. A lesion revealed on X-rays was diagnosed as an osteochondroma of the medial border of his scapula. After preoperative examinations, he underwent open surgery in order to remove the lesion. A histological examination confirmed the clinical diagnosis of osteochondroma. A clinical examination 3 months later showed a full and painless range of motion, the absence of the grating sensation during passive abduction and elevation and the complete disappearance of his left shoulder deformity. After 2 years of follow-up, there were no clinical or radiological signs of recurrence.

Conclusions: Despite its rarity osteochondroma should be considered in the differential diagnosis for any adolescent presenting with a winging of the scapula.

Keywords: Adolescents, Benign tumors, Osteochondroma, Pseudowinging, Winged scapula





Conclusions

Scapular Winging is a rare, potentially debilitating disorder with many causative factors.

Diagnosis is largely clinical and relies on a large number of different suspects.

A common pitfall is the failure to undress the patient's shoulders and back to the waistline, which permits adequate visualization of any obvious deformity.

Early diagnosis, rehabilitation and, in some cases, surgical intervention improve the outcomes of patients

European PCmpe Consortium

- Prof. A. van der Ploeg, The Netherlands
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UOC Neurologia e Malattie Neuromuscolari

G. Vita
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C. Rodolico
A. Mazzeo
O. Musumeci
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