See it, think it, test it:
herpes-like eschar in ulceroglandular tularemia

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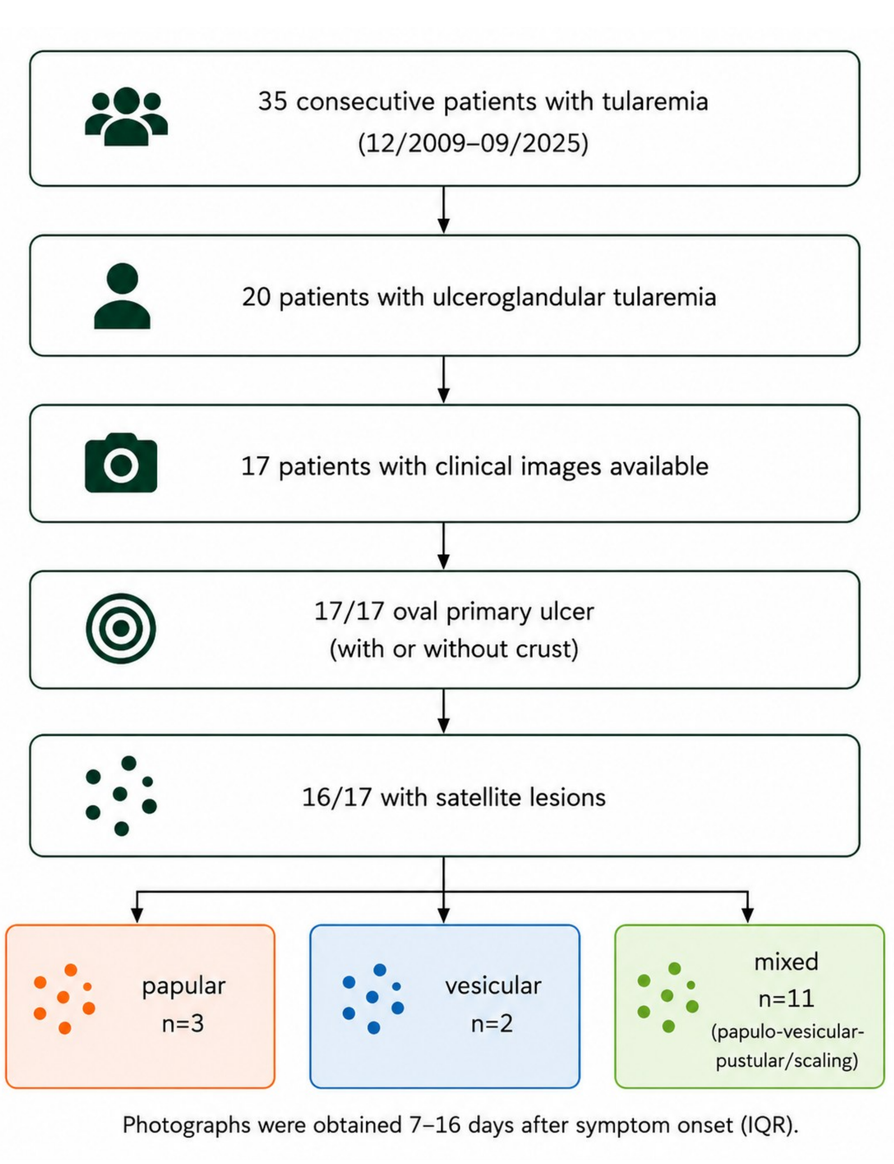
BACKGROUND

- Tularemia is a rare but highly virulent zoonotic, arthropod-borne disease caused by *Francisella tularensis*.
- In Central Europe, its most common pediatric presentation is ulceroglandular = eschar (i.e., an entry-site ulcer) + regional lymphadenitis.
- Localized skin lesions near inflamed lymph nodes are common in children and may be unrelated.
- Based on our earlier observations[1] and inspired by a case reported by Byington et al. in 2008[2], **we reviewed the appearance of all eschars in microbiologically confirmed tularemia cases at our Central Europe institution** for which photographic documentation was available.

DISCUSSION

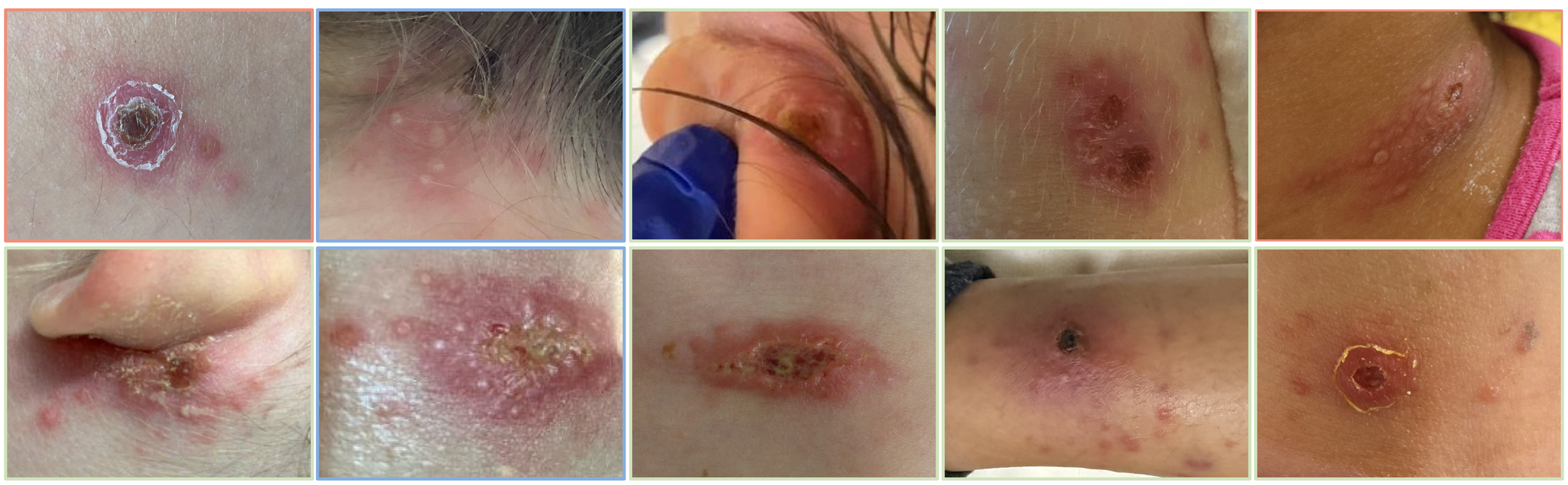
- The **presence of these herpes-like lesions surrounding an eschar**, in combination with acute regional lymphadenitis, should raise clinical suspicion for tularemia.
- Recognition of this characteristic pattern may justify:
  - early microbiological testing (rapid antibody screening/PCR from eschar)
  - initiation of intracellularly active antibiotic therapy (ciprofloxacin or doxycycline)
- Differential diagnoses associated with eschars (e.g., bacterial ecthyma, rickettsial infections) typically lack satellite lesions.
- Not all satellite eruptions are necessarily pathogen-related, as some may reflect secondary irritative changes caused by wound dressings or topical therapies.
- The histomorphologic pattern of these satellite lesions remains unclear. Whether they contain *F. tularensis* DNA may be of interest for future research.

CASE PRESENTATION



Photographs were obtained 7–16 days after symptom onset (IQR).

CLINICAL IMAGES



TAKE-HOME MESSAGE

SEE IT
Herpes-like satellite lesions around an eschar

THINK IT
Ulceroglandular tularemia

TEST IT
Serology and/or PCR

TREAT IT
Early intracellularly active antibiotics

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From Head to Toe: Diagnostic Challenges in Guillain-Barré Syndrome with Miller-Fisher Features

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BACKGROUND

- Guillain-Barré syndrome (GBS) is an acute inflammatory polyradiculoneuropathy typically triggered by infection.
- It is usually characterized by ascending weakness from lower to upper limbs and cranial muscles.
- GBS may overlap with a variant of this acute inflammatory neuropathy, the Miller-Fisher syndrome (MFS) with its triad of 1) ataxia, 2) areflexia, and 3) ophthalmoplegia.

DIAGNOSIS

- Primary: Clinical findings
- Supportive:
  - •Cerebrospinal fluid (CSF): albuminocytological dissociation
  - •Electrophysiology (ENMG): signs of acute demyelinating or axonal lesions
  - •MFS: Anti-GQ1b antibodies are detected in approximately 80% of patients
  - •Neuroimaging: normal or thickening and contrast enhancement of the spinal nerve roots and cranial nerves. Important to exclude differential diagnoses

DIFFERENTIAL DIAGNOSIS

- Cerebral or cerebellar inflammation, infections, toxic, metabolic, or structural disorders of the central nervous system

TREATMENT

- Intravenous immunoglobulins (IvIG) or plasma exchange
- The outcome is generally good; residual deficits are rare

Fig 1. Clinical presentation: Bilateral abducens palsy: here left-sided esotropia, compensatory head-tilt and eye-cover
Photograph with kind permission of family

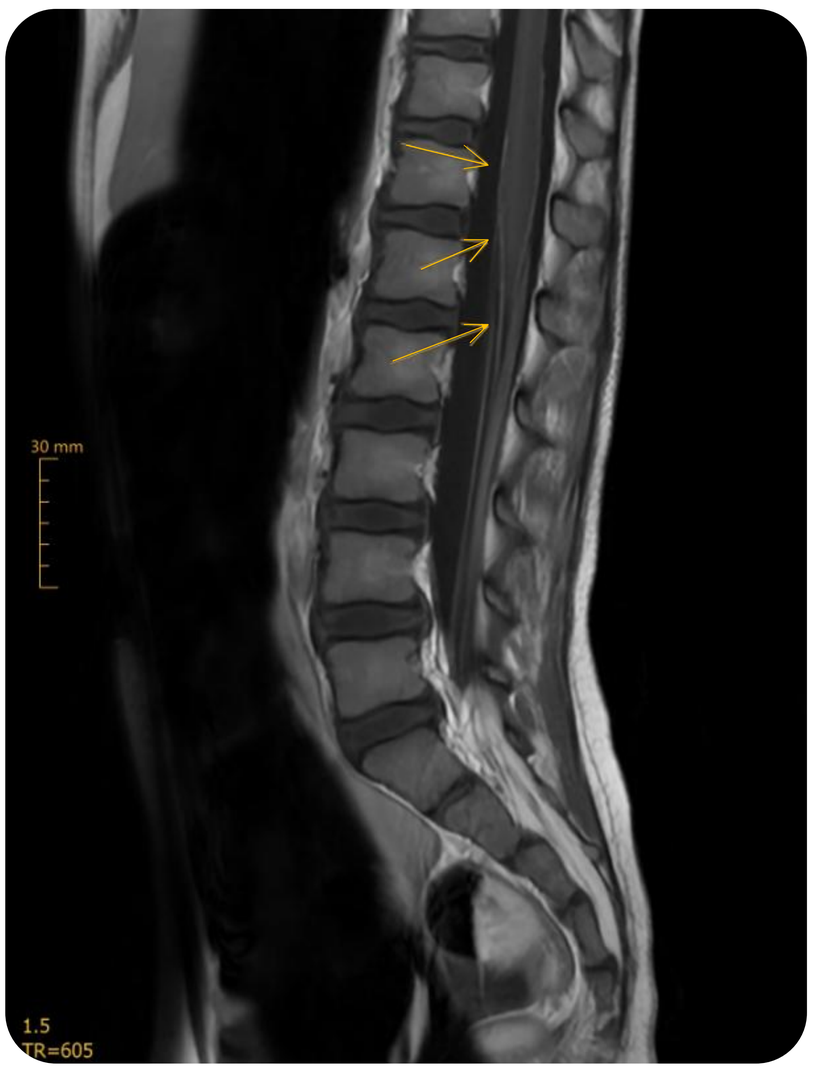

Fig 2. MRI (contrast enhanced T1 sagittal) showing cauda equina enhancement consistent with radiculitis

CASE REPORT

- 6-year-old girl with a 3-day history of strabismus, headache, and morning vomiting
- Clinical findings: bilateral abducens and oculomotor nerve palsy
- Head CT-Scan and CSF: initially normal
- Admission to pediatric ward; neurological deterioration with progressive weakness of extremities, areflexia, dysphagia, and incomplete ophthalmoplegia
- Transfer to an intensive care unit and start IvIG therapy based on suspicion of GBS/MFS
- Clinical improvement and positive response of neuropathic pain to gabapentin
- Atypical for GBS: during recovery development of right-sided asymmetry
- Atypical for MFS: anti-GQ1b antibody remain negative
- Diagnostic confidence through:
  - •CSF: second puncture: albuminocytological dissociation
  - •Spinal MRI: cauda equina enhancement consistent with radiculitis
  - •Neurography: absent F-waves and present A-waves (indicative of acute demyelination)
  - •Response to IvIG treatment
- Diagnostic workup: acute Epstein-Barr virus infection as the potential trigger. In EBV triggered GBS typically anti-GQ1b antibodies remain negative
- Transfer to pediatric neurological rehabilitation center: substantial recovery

CONCLUSION

Guillain-Barré Syndrome and its Miller-Fisher variant are rare in pediatric patients and show atypical or evolving presentations.

Diagnosis is primarily clinical as supportive diagnostic findings may be delayed. Close follow-up is important.

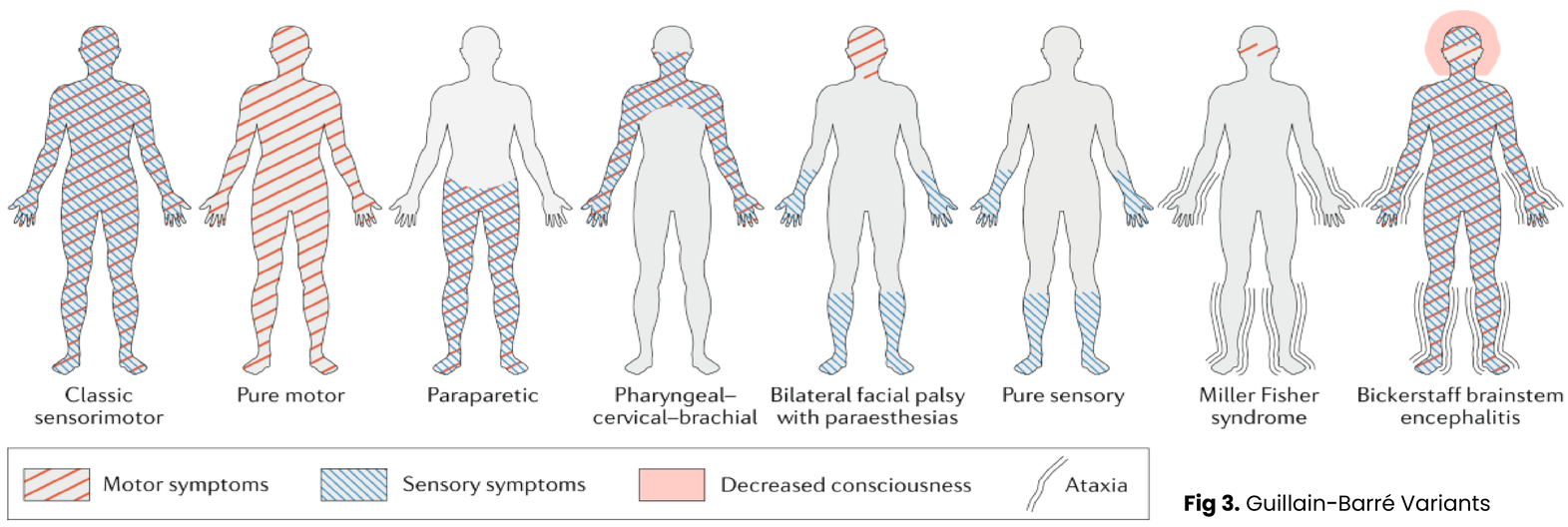

Fig 3. Guillain-Barré Variants

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When Glucose and Electrolytes don't tell the Same Story

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Introduction

- **Type 2 diabetes** is increasingly diagnosed in adolescents
- Electrolyte disturbances are **not typical** of type 2 diabetes
- **Unexpected abnormalities** should trigger further investigations
- Initial diabetes work-up led to a **suprising diagnosis**

Case Presentation & Work-

Patient presentation

- **11 year old adolescent girl**
- **Hyperglycaemia** discovered on outpatient testing
- Chronic **polydipsia** + **fatigue**
- Mild obesity (BMI 28.24kg/m2)
- Acanthosis nigricans
- Family history: T2DM
- Parental consanguinity

First investigation

HbA1c	**7.1 %**
Glycaemia	**16,9 mmol/l**

Diagnosis

Newly diagnosed Type 2 diabetes mellitus

Unexpected findings

Findings not explained by T2DM Laboratory results:

- **K+ 2.2 mmol/l**
- **Mg 0.49mmol/l**
- **Metabolic alkalosis**
- **Hypochloraemia**

Further investigations

- Renal salt wasting
- Tubular reabsorbtion studies
- Distal convoluted tubule defect

Diagnosis

GITELMAN SYNDROME

Treatment

- Electrolyte supplementation.
- Diabetes management
- Multidisciplinary follow up

Discussion

Electrolyte disturbances are not typical in T2DM

Severe hypokalaemia and hypomagnesaemia should prompt complementary investigations for associated disorders.

Interaction between diabetes and Gitelman syndrome

Chronic hypokalaemia and hypomagnesaemia may worsen insulin resistance and glucose metabolism.

Combined management

Early recognition allows optimization of both electrolyte and glycaemic control.

Silent Deficiency, Visible Deformity: A Case Report of Severe Nutritional Rickets in a Toddler

KSA Kinderspital Aarau

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Background

- Rickets = skeletal disorder of bone mineralization
  - soft bones, bone deformities, growth impairment
- Caused by vitamin D, calcium, or phosphate deficiency
- Risk factors for nutritional rickets
  - ➢ Exclusively breastfed infants due to the low vitamin D content of breast milk
  - ➢ Darker skin
  - ➢ Born prematurely
  - ➢ Living in regions with low sun exposure

Case Description

History of Present Illness: 2.5-year-old girl with progressive bowing of the legs since early infancy. Full-term birth, exclusively breastfed, no vitamin D or dairy intake.

Physical Exam: Bilateral genu varum, thickened wrists/ankles, height 79 cm (<3rd percentile) were noted (Figure 1a,b).

Laboratory Study:

- ↓ 25-hydroxyvitamin D (19.9 nmol/L, ref: 50-250 nmol/L)
- ↑↑ 1,25-dihydroxyvitamin D (>480 pmol/L, ref: <363.0 pmol/L) [a]
- ↓ calcium (1.97 mmol/L, ref.: 2.15-2.6 mmol/L)
- ↓ phosphate (0.97 mmol/L, ref: 1-1.8 mmol/L)
- ↑ parathyroid hormone (743 ng/L, ref.: 15-68.3 ng/L)
- ↑ alkaline phosphatase (1555 IU/L, ref.: 191-450 IU/L)
- Normal renal function and thyroid, and no phosphate wasting

Imaging: Radiographic imaging revealed **epiphyseal cupping** and severe osteopenia (Figure 2).

Therapy: Oral cholecalciferol 4,000 IU/day for 4 months, tapered to 400 IU/day, and encouraged dairy intake over 2 years. Gradual correction of bone deformities, normalized lab values, and full clinical recovery (Figure 1c and 2c).

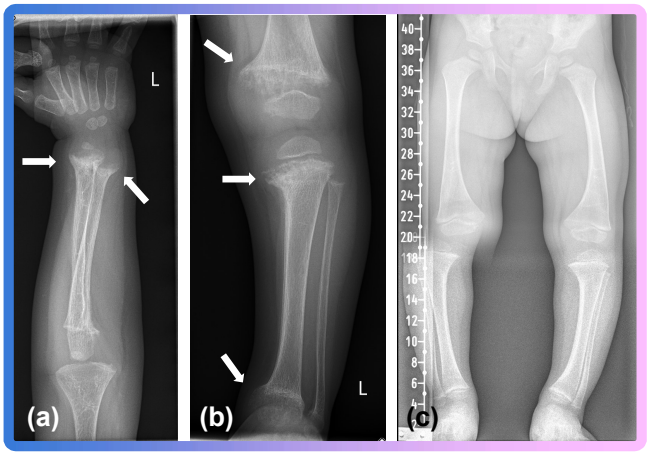

Figure 2: (a) Radiographs of the left arm and (b) left lower leg taken at the initial visit, showing cupping (white arrows). (c) Follow-up radiograph of the lower extremities after 6 months, showing regression of the typical rachitic bone changes and, in comparison to the photographs in Figure 1, an improvement in leg alignment.

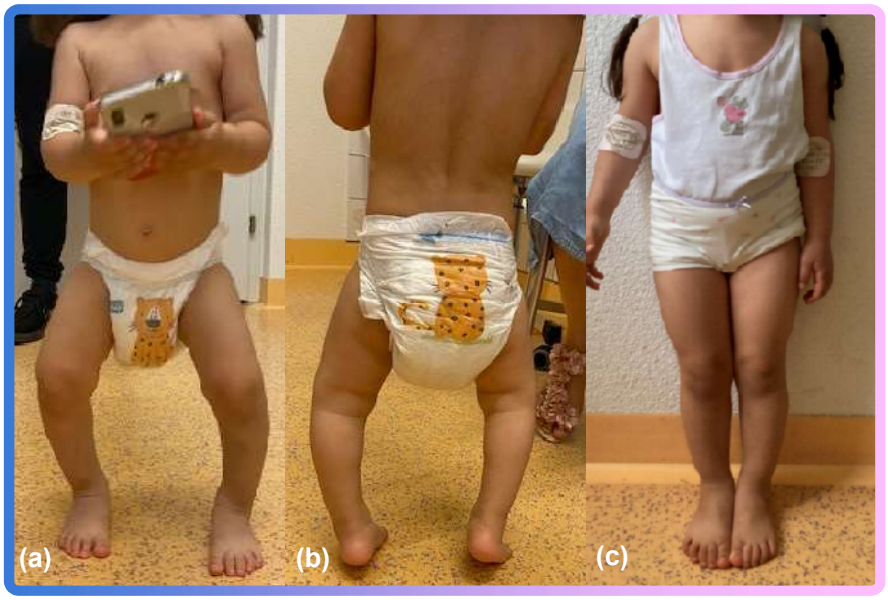

Figure 1: Partial-body photographs from the initial presentation to the ED showing (a) a frontal view and (b) a dorsal view, both demonstrating severe bilateral genu varum. (c) A follow-up frontal view taken after two years illustrates significant improvement in leg alignment.

Diagnostics

Clinical:

- Infants: craniotabes (cranial softening), delayed fontanel closure
- Older children: genu varum/valgum, rachitic rosary (swelling of anterior rib ends), cupping (swelling of wrists or ankles), poor growth
- In severe cases: failure to thrive, lethargy, hypocalcemic seizures, and anemia

Laboratory:

- ↓25-hydroxyvitamin D, hypocalcemia, hypophosphatemia, ↑ parathyroid hormone and alkaline phosphatase activity
- Early stage 1,25-dihydroxyvitamin D concentrations may be elevated in response to hypocalcemia, but progressive substrate depletion results in subsequent reductions in later stages

Radiology: Cupping, fraying, and osteopenia at metaphyses

Management

Oral cholecalciferol supplementation:

- Intensive phase: 2'000–10'000 IU/day for up to 12 weeks
- Maintenance phase: 400–1000 IU/day for 3-4 months
- Alternative: single dose (Stoss therapy) of up to 600'000 IU intramuscularly

Calcium: 500–1,000 mg/day through diet to prevent post-treatment hypocalcemia

Key points

- Vitamin D supplementation (recommendation 400 IU daily from birth to 12 months, followed by 600 IU daily until 3 years) in infants is important to prevent rickets, especially exclusively breastfed, premature born, or dark-skinned infants.
- Early recognition and treatment of nutritional rickets usually results in complete recovery. Pediatricians and endocrinologists play a crucial role in prevention and management.

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Benign but Misleading: A Case of Van Neck-Odelberg Disease Masquerading as Malignancy

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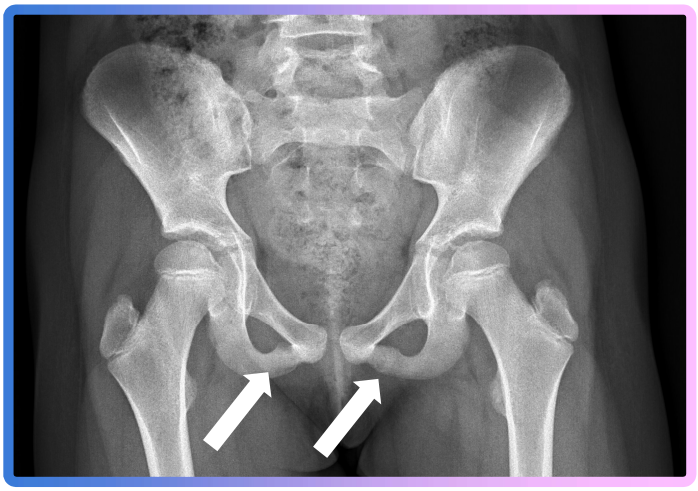


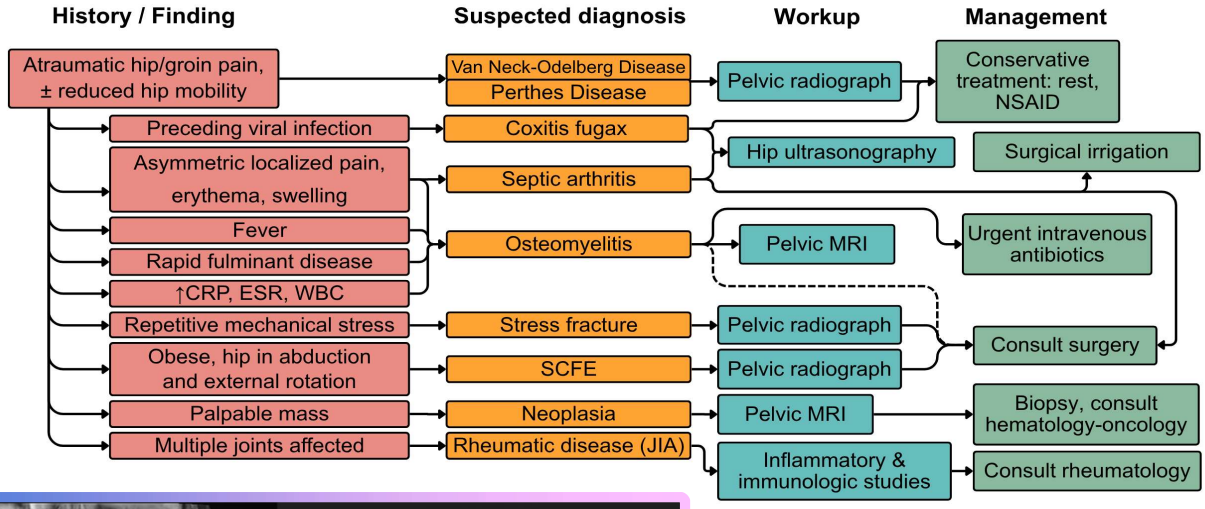
Figure 1: anterior-posterior pelvis radiograph showing slight **enlargement** on both ischiopubic synchondroses with **osteolytic** appearances on the left side.

Case Description

- **History of present illness:** 9-year-old girl, acute atraumatic right-sided hip pain and limping gait, no fever or relevant medical history.
- **Physical exam:** ↓right hip abduction, flexion, and internal rotation
- **Laboratory study was normal**, including full blood count, inflammatory studies, bone metabolism, immunologic parameters
- **Imaging:** Pelvic radiograph and MRI: Figure 1 and 2.
- **Therapy:** Physiotherapy, rest, NSAID

Management

- Excellent prognosis with **NSAID** and **rest.**
- In persistent cases: radiographic follow-up and re-evaluation of differential diagnoses.



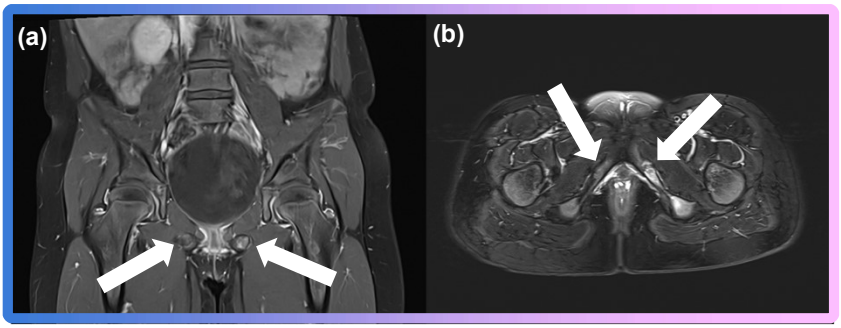


Figure 2: MRI T2-weighted sequences (a: coronal view, b: axial view) revealing slight bilateral enlargement of the ischiopubic synchondrosis with left-sided edema and contrast enhancement without any soft tissue involvement (white arrows).

Background

- Van Neck–Odelberg disease (VND) = symptomatic fusion of the ischiopubic synchondrosis (IPS)
- **Benign,** self-limiting form of skeletal maturation
- Often asymmetrical and related to leg dominance
- Atraumatic hip pain, reduced hip mobility, limping
- Clinical presentation and non-specific imaging features can mimic osteomyelitis, stress fractures, or malignant bone tumors, posing a diagnostic challenge.

Key points

- VND should be considered in children presenting with atraumatic hip pain and suspicious IPS imaging findings.
- Diagnostic workup includes pelvic imaging with X-ray and MRI, with inflammatory, immunologic, and bone metabolism studies.
- Conservative management of VND has an excellent prognosis.

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CASE REPORT · SWISS SOCIETY OF PEDIATRICS 2026

Persisting Acute Cerebellar Ataxia in Infancy
A Rare Presentation of Neuroblastoma

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Departement of Woman–Mother–Child

INTRODUCTION

- Acute cerebellar ataxia accounts for 1 in 5,000 paediatric emergency department attendances
- Most cases are post-infectious and self-limited
- When ataxia persists beyond 2 weeks without fever, trauma, or recent infection, a serious underlying aetiology must be actively sought

Opsoclonus Myoclonus Ataxia Syndrome (OMAS) associated with neuroblastoma in children under 3 years is classically recognised by its clinical triad
Ataxia can be the sole initial manifestation

CLINICAL PRESENTATION

PATIENT
13-month-old girl, no prior medical history

Onset
Subacute onset over 2 weeks

GAIT
Progressive instability enlarged lift polygon

No red flags
No fever, no trauma, no infection, no toxic

VITAL SIGNS
Normal, No fever

TREMOR
Action tremor both arms + head tremor

TRUNCAL ATAXIA
Present, prominent

NEURO EXAM
No opsoclonus, no myoclonus

DIAGNOSTIC JOURNEY

1 *Admission*

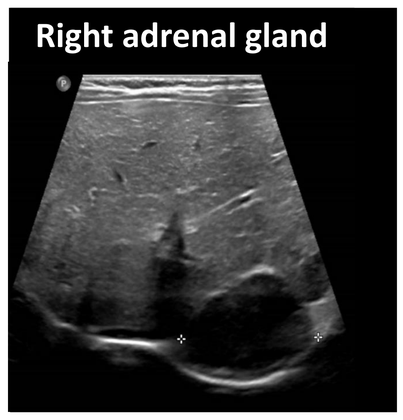
Blood tests + non-contrast brain MRI → unremarkable
Post-infectious ACA initially considered

2 *Day 7 — symptoms persist*

Persistent ataxia without infection or trauma → paraneoplastic syndrome considered

3 *Abdominal ultrasound*

Heterogeneous, vascularized right adrenal mass → neuroblastoma suspected



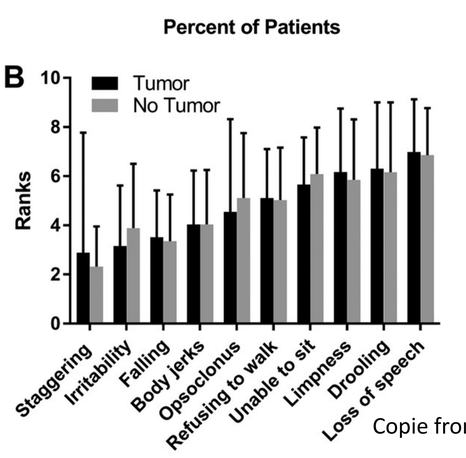
4 Transfer to oncology + Treatment

No metastatic disease.
Exclude mimics with PL
Surgical resection of neuroblastoma + corticosteroids
Neurological improvement

DISCUSSION

Pathophysiology

OMAS is an immune-mediated paraneoplastic disorder involving cerebellar and brainstem circuits
Neuroblastoma triggers an immune response cross-reacting with neural antigens, causing cerebellar dysfunction even before opsoclonus and myoclonus appear



Copie from Pranzatelli et al 2018

Why the Diagnosis Was Delayed ?

Misleading features

Normal brain MRI
Paraneoplastic ataxia causes no structural lesions on standard MRI

Incomplete OMAS triad
Isolated ataxia does not trigger the classic OMAS clinical alarm

TAKE-HOME MESSAGE

Ataxia lasting 2 weeks < 2 years old = uncommon presentation → think of neuroblastoma

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Unwitnessed Button Battery Ingestion in a 16-Month-Old Girl: A Hidden Danger With Serious Consequences

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Background

Button batteries pose a risk of accidental ingestion among children, particularly for those under six years (1). Diagnosis of unwitnessed pediatric button battery ingestion is challenging. Non-specific symptoms can lead to misdiagnosis with delayed removal and increased risk of complications (2). We present the clinical course, diagnosis, and management of a toddler with an unwitnessed button battery ingestion, presenting with chronic dysphagia.

Case Presentation

Emergency Department

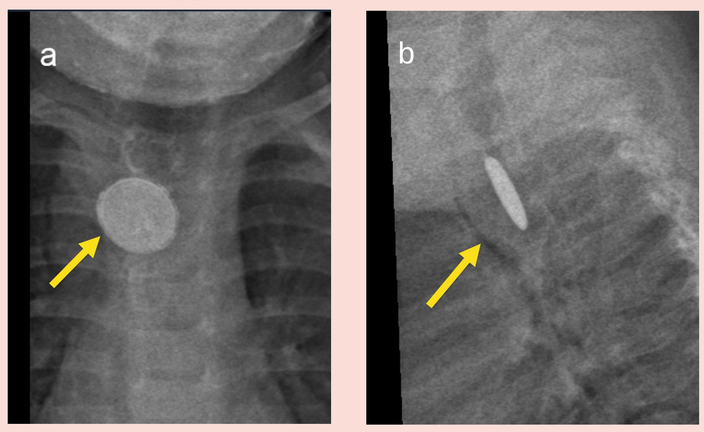
- 16-month-old girl
- Vomiting after each meal for ten days, no diarrhea, no fever
- Mild leukocytosis (13.78 G/l), low CRP (2 mg/l),
- known microcytic hypochromic anemia (Hb 78 g/l)
- Hospitalization for poor oral intake
- Suspected infectious etiology

Ward

- Five-month history of feeding difficulties and refusal of solids, beginning with solid food introduction
- Repeated swallowing attempts, retching, and vomiting directly after food intake, subsequent inspiratory stridor
- Diagnosis of chronic dysphagia

Diagnostic work-up

Fluoroscopy



Suspicion: Unwitnessed button battery ingestion with unclear duration of impaction

Figure 1: Initial fluoroscopy of the esophagus
a) Anteroposterior view: A round, metallic-dense foreign body with a double-ring sign projected over the proximal esophagus (arrow).
b) Lateral view: Adjacent edema, tracheal displacement, and filiform narrowing (arrow).

Contrast-enhanced computed tomography (CT)

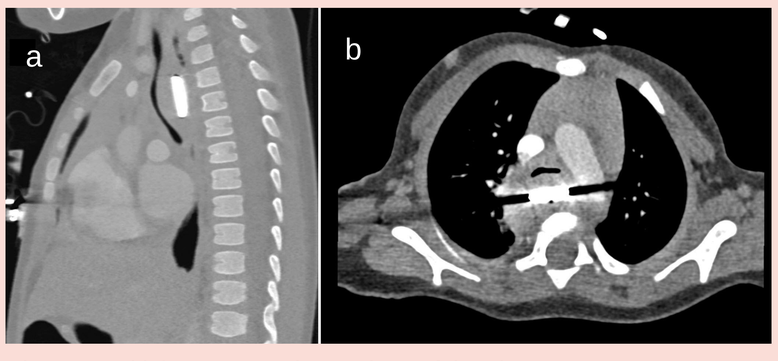
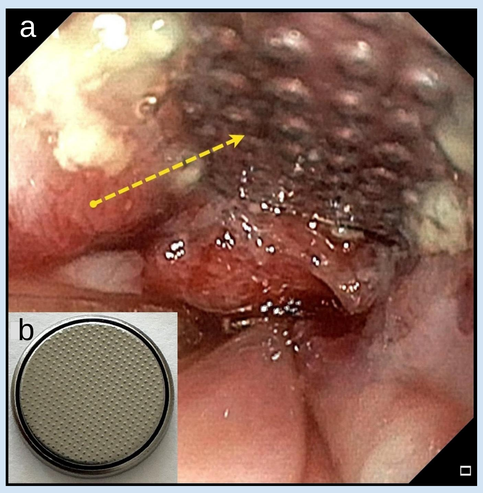


Figure 2: Initial contrast-enhanced CT of the chest
a) Sagittal view: Marked periesophageal soft tissue swelling with consequent tracheal displacement and focal narrowing.
b) Axial view: No evidence of esophago-tracheal or vascular fistula or abscess collection.

Management

Esophagoscopy



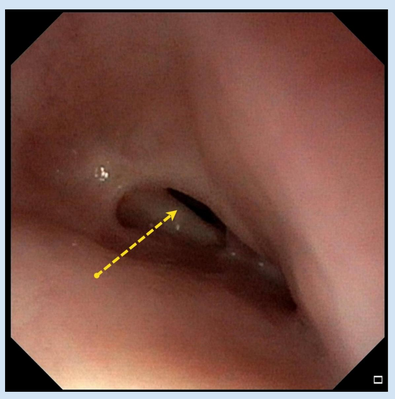
- Impacted foreign body in a pocket-like structure in the proximal esophagus with consecutive luminal narrowing
- Difficult removal after several attempts using rigid esophagoscopy

Identification of the foreign body as a 20 mm button battery

Figure 3:
a) Endoscopic view showing the back of a lodged button battery (arrow) in the proximal esophagus prior to removal.
b) Exemplary photograph of the back of a CR2032 BB with punctate surface markings.

Complication

Esophageal stricture



- Development of an esophageal stricture at the former button battery location, requiring three balloon dilations

Figure 4:
Endoscopic view ten months after button battery removal, demonstrating sail-shaped esophageal narrowing (arrow) beneath a pocket-shaped enlargement at the former button battery location.

Conclusions

Unwitnessed button battery ingestion can present with subtle and fluctuating aerodigestive symptoms that can mimic benign conditions.

Our case highlights the presence of dysphagia as a warning sign for foreign-body related swallowing impairment.

Persistent or unexplained dysphagia must be systematically investigated, as delayed diagnosis of button battery ingestion may result in serious and potentially fatal complications.

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INFLUENZA'S CHERRY ON TOP

CASE

A HEALTHY VACCINATED 8-MONTH-OLD BOY WITH INFLUENZA A AND GASTROINTESTINAL PREDOMINANCE WHO RAPIDLY DETERIORATED INTO LIFE-THREATENING ACUTE EPIGLOTTITIS.

DAY

0 — HOME
ONSET OF FEVER, COUGH AND RHINORRHEA

2 — EMERGENCY DEPARTMENT
DIAGNOSIS OF UPPER RESPIRATORY TRACT INFECTION AND DISCHARGED HOME

4 — PEDIATRICIAN
FEVER, VOMITING, WATERY DIARRHEA, DECREASED ORAL INTAKE

TRANSFERED BACK TO THE ED BY THE PEDIATRICIAN ON DAY 4

☐ PHYSICAL EXAMINATION
- ILL, IRRITABLE, FEBRILE, TACHYCARDIC, NORMAL BP AND MODERATE DEHYDRATION
- MILD COUGH. ERYTHEMATOUS OROPHARYNX WITH BILATERAL TONSILLAR HYPERTROPHY

☐ LABORATORY TESTING
- RAPID TESTING POSITIVE FOR INFLUENZA A
- CBC, CRP, ELECTROLYTES, RENAL AND HEPATIC FUNCTION WITHIN NORMAL RANGE.

☐ PLAN
- PRESUMED DIAGNOSIS : INFECTIOUS GASTROENTERITIS IN THE CONTEXT OF INFLUENZA A
- HOSPITALISATION FOR IV REHYDRATION AND OSELTAMIVIR THERAPY

6 — PEDIATRIC WARD
ASTHENIC, HOARSE VOICE, LARYNGEAL COUGH (NO STRIDOR) : ORAL DOSE OF DEXAMÉTHASONE.
PERSISTENT APATHY AND APPARENT PHOTOPHOBIA : LUMBAR PUNCTURE AND INITIATION OF MENINGITIS-DOSE CEFTRIAXONE

7 — PEDIATRIC INTENSIVE CARE UNIT
ACUTE RESPIRATORY DETERIORATION

☐ PHYSICAL EXAMINATION
- INSPIRATORY STRIDOR, DROOLING, RESPIRATORY DISTRESS, NECK KEPT IN HYPEREXTENSION

☐ NASOFIBROSCOPY
- EPIGLOTTITIS WITHOUT ABCESS FORMATION, PRESERVED AIRWAY PATENCY

☐ PLAN
- URGENT ENDOTRACHEAL INTUBATION
- TRANSFER TO PICU

10 — PEDIATRIC INTENSIVE CARE UNIT
GRADUATE CLINICAL IMPROVEMENT LEADING TO EXTUBATION
HYPERACTIVE DELIRIUM REQUIRING ONGOING SEDATION

13 — PEDIATRIC WARD
2ND NASOFIBROSCOPY: RESIDUAL MILLIMETRIC GAP DURING ADDUCTION OF THE LEFT VOCAL CORD.
REINTRODUCTION OF ORAL FEEDING
PROGRESSIVE IMPROVEMENT

20 — HOME
DISCHARGED HOME

47
FULL RECOVERY AT ENT 1 MONTHS CHECK-UP

TAKE HOME MESSAGES

- VACCINATION DOES NOT RULE OUT EPIGLOTTITIS. ALWAYS KEEP THIS DIAGNOSIS IN MIND IN ANY CHILD PRESENTING WITH STRIDOR, EVEN IF THE CHILD IS FULLY VACCINATED.
- GASTROINTESTINAL SYMPTOMS ARE AN ATYPICAL BUT POSSIBLE PRESENTATION OF INFLUENZA A INFECTION
- CLOSE MONITORING AND REPEATED REASSESSMENT IS NECESSARY TO DETECT AIRWAY COMPROMISE EARLY, EVEN IN INITIALLY STABLE PATIENTS

Hôpitaux Universitaires Genève

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REFERENCES & CONTACTS

FROM GLASSES TO AZATHIOPRINE: THE CHALLENGING DIAGNOSIS OF JUVENILE MYASTHENIA GRAVIS

Marine Dumas [a] / Alain Lefèvre-Utile [a] / David Jacquier [b] / Frida Rizzati [c] / Alexis Rybak [d] / Ophélie de Pury [d]

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1 - ABSTRACT

Juvenile myasthenia gravis (JMG) is a rare autoimmune disorder affecting the neuromuscular junction, rarely seen in childhood. In toddlers, heterogeneous clinical presentations and fluctuating symptoms often make early diagnosis challenging. We report the case of a young child who developed a severe complication of previously unrecognized JMG, highlighting the importance of considering this diagnosis in cases of fluctuating weakness or other heterogenous, progressive manifestations.

2 - MEDICAL HISTORY

Previously healthy 2-year-old girl presents repeatedly to the pediatric emergency department with:

- Two-month history of fluctuant, but overall progressive regression of motor, social and verbal skills
- Increased frequency of falls
- Newly developed left-sided ptosis with divergent strabismus. Wearing corrective glasses prescribed by an ophthalmologist
- Food selectivity and chewing/swallowing difficulties

3 - CLINICAL EXAMINATION

Neurological :

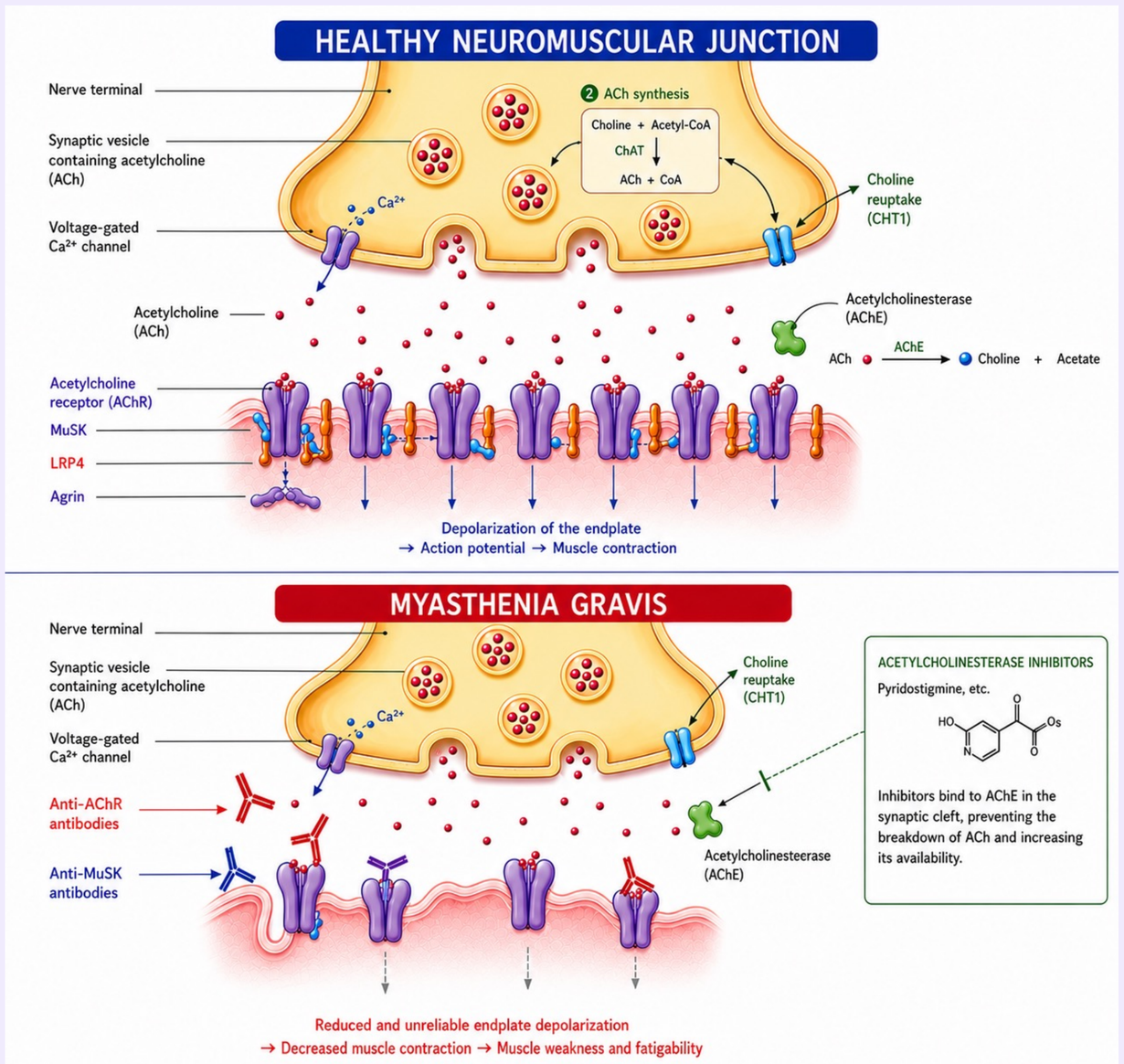
- Generalized hypoactivity and fatigability
- Facial hypomimia
- Bilateral ptosis, left divergent strabismus; normal eye movement
- Poor social interaction; no verbal interactions

➢ **Marked variability between examinations !!**

ENT : rhinorrhea and blocked nose, cough

Remainder of physical examination: normal

4 - PHYSIOPATHOLOGY



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5 - DIAGNOSTIC WORK-UP

- **Metabolic screening:** unremarkable
- **Brain and spine MRI:** normal
- **Multidisciplinary discussions** with specialists...

... JMG = clinical diagnosis !! After initial clinical suspicion:

- **Electroneuromyography (ENMG):** pathological decrement on repetitive nerve stimulation
- **Serum anti-acetylcholine receptor antibodies:** positive
- **Thoracic MRI:** no thymoma

➢ **Therapeutic and diagnostic trial with acetylcholinesterase inhibitors (AChEi)**

6 - THERAPY INITIATION AND FOLLOW-UP

Post- ENMG results, transfer to ICU for **AChEi trial**: potential life-threatening side effects, clinical worsening in genetic congenital myasthenic syndromes

↓

Multifactorial acute respiratory failure: pneumonia, **myasthenic crisis** (serum anti-acetylcholine receptor antibodies positive)

↓

Treatment of severe forms of JMG:
Intraveinous immunoglobulins, systemic corticoids, non-invasive ventilation

↓

Rapid clinical improvement

↓

Long-term follow-up: switch to relay therapy (azathioprine)
Phasing out of corticoids

↓

At 11 months of history: patient free of any therapy, healed

7 - TAKE-HOME MESSAGES

Juvenile myasthenia gravis = a challengig diagnosis in children.

- **Fluctuant and progressive** ocular and/or bulbar symptoms
- Misleading and **heterogenous** early symptoms falsely attributed to benign ophthalmological conditions. Subtle or absent limb weakness
- Importance of early recognition to prevent life-threatening complications such as myasthenic crisis
- **Clinical diagnosis**! Supporting exams are ENMG and response to acetylcholinesterase inhibitors (AChEis)

  Beware: AChEis may cause clinical worsening in some genetic congenital myasthenic syndromes.

Nursing Strategies and Tools to Support Treatment Adherence in Adolescents With Asthma

Syrine Merrouche, BSc, RN ; Imane Safi, BSc, RN ; Jenesa Srikantha, BSc, RN

Supervised by Frédérique Debbiche, MSc, RN

1 INTRODUCTION

CONTEXT

11% of adolescents are affected by asthma [1] and only 1 in 2 adolescents regularly adheres to their treatment [2].

Adolescence = a high-risk period

- Quest for autonomy
- Peer influence
- Behavioural variability

Consequences of non-adherence

- Exacerbations and hospitalizations
- Reduced quality of life
- High healthcare system costs

OBJECTIVES

To identify factors influencing treatment adherence and propose evidence-based nursing strategies.

Guided by Meleis' Transition Theory [3], this work focuses on :

- The adolescent's developmental transition
- The health–illness transition
- The transition toward autonomy in care management

2 METHODS

454 articles identified in the PubMed database

→ **61** articles excluded due to :
- General chronic conditions
- Non-relevant age group
- Inappropriate study design
- Limited publication dates

74 articles identified

→ **4** articles excluded due to :
- Hospital setting vs. school setting
- Non-relevant results
- Not directly related to nursing care

13 articles eligible for analysis

9 articles included in the analysis

Search period : 2015 – 2025

Database : PubMed

Target population : Adolescents 12–20 years

IMRD-structured articles

3 RESULTS [4-12]

BARRIERS

- Trivialisation of symptoms
- Limited knowledge of the condition
- Misconceptions about treatments
- Medication non-adherence / missed doses
- Stigmatisation

FACILITATORS

- Therapeutic education
- Motivational interviewing
- Family support
- Tailored communication

TRANSITION

The transition to independence remains insufficiently structured.
Adolescents want to be involved in their care but often feel ill-prepared to manage their condition independently.

NURSING STRATEGIES

- Gradual involvement
- Appointments without parents
- Personalised goals
- Ongoing support

THE ROLE OF A NURSE

The nurse acts as an educator, mediator, source of psychosocial support, and facilitator of transition.

DIGITAL HEALTH TOOLS

Digital tools improve adherence, self-monitoring, and a sense of control.
Digital tools identified : mobile apps , smart inhalers, interactive questionnaires, remote monitoring

Paul, a 15-Year-Old Adolescent : Supporting the Adolescent Transition Toward Autonomy

Identifying key stages of the adolescent care pathway and interventions promoting self-management

Arrival at Pediatric Emergency Department
- Assessment of transition stage
- Semi-structured *HEADSSS* interview
- **Nursing pocket guide**

Hospitalization in Pediatrics
- Therapeutic patient education (ETP)
- Personalized action plan
- **Nursing transition protocol**
- Testimonial video → Mirror effect → Promotion of autonomy

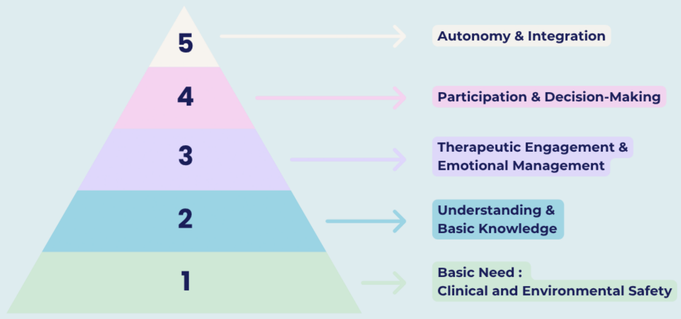
Preparation for Return Home
- Parent liaisonform
- **Adolescent checklist**
- Concrets goals plan
- Care network

Return Home and Outpatient Follow-up
- Assess progression of autonomy
- Remote nursing follow-up
- Self-monitoring booklet
- **"Asthma and Me" kit**

4 DISCUSSION & RECOMMENDATIONS

STEPWISE MODEL OF AUTONOMY DEVELOPMENT

Inspired by Maslow's hierarchy of needs and Meleis' Transition Theory



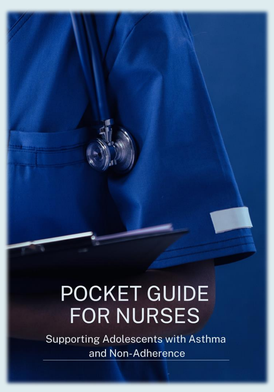
NURSING STRATEGIES

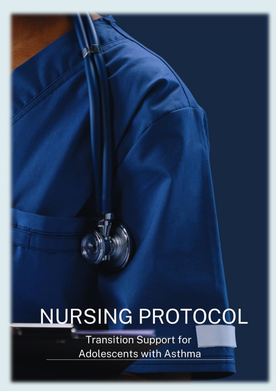
- Identification of non-adherence risk factors
- Structured nursing assessment
- Individualized therapeutic education
- Support for autonomy
- Integration of psychosocial dimensions
- Support for adolescent-to-adult transition
- Motivational interviewing

EDUCATIONAL KIT "ASTHMA & ME"

- Card Game : True or False about Asthma
- My Asthma Story
- Understanding Asthma : Video Support
- Quiz : My Asthma and Me

3 NURSING TOOLS





5 CONCLUSION

Nurses play a key role in supporting adolescents throughout the transition toward autonomous disease management.

The findings of this work will also be published in *Soins Pédiatrie-Puériculture*.

" Empowered Today, Autonomous Tomorrow. "

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Explore Tools & Resources !

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Pädiatrie Schweiz · 11–12 juin 2026

Département femme-mère-enfant
Pneumologie et mucoviscidose

Beware the Dormant Water:

Immune-Mediated Cardiac Complications of C. jejuni Infection

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CLINICAL TIMELINE

Prior History — Elite skier, mild exercise induced asthma

***C. Jejuni* infection** — Severe GI illness, fever, dehydration, malaise after ingesting contaminated water

Recovery

Symptom Onset — Exertional dyspnea, palpitations after exercise

Work-up

Cardiac MRI — > 3 months post-onset: No active inflammation, No scar

Return to Play

CASE REPORT

Patient — Elite Female Skier

Background: Mild exercise-induced asthma. No prior cardiac history.

Presenting complaint: New onset exertional dyspnea described as inspiratory-expiratory "chest obstruction", distinct from prior asthma flares. Palpitations upon exercise cessation — extra flutters triggering cough.

Antecedent illness: Recent hospitalisation for *C. jejuni* infection (contaminated water source) — severe gastrointestinal symptoms, fever, dehydration and malaise.

Symptom onset: Upon resuming training after 3-week recovery period. No symptoms during rest.

Physical Examination & Vital Signs

→ Unremarkable

INVESTIGATIONS

Spirometry Mild obstructive pattern
FEV_1/FVC Z-score −2.27
Nonsignificant bronchodilator response (FEV_1 +9.9%)

ECG Sinus rhythm
Normal QRS axis and intervals
No repolarisation abnormalities

Transthoracic Echocardiography
Normal biventricular structure and function
No valvular pathology

Maximal CPET
VO_2 peak: 39 ml/kg/min (120% predicted), peak HR: 201/'
No arrhythmia during incremental exercise
Frequent VES + 3-beat NSVT during recovery

Cardiac MRI (>3 months post-onset)
No active inflammation
Normal biventricular structure and function
No myocardial scar (LGE negative)

MANAGEMENT & OUTCOME

Presumptive diagnosis: Post-infectious myocarditis

Low-dose long-acting metoprolol → initiated as anti-arrhythmic strategy

Cardiac MRI (deferred > 3 months) → no inflammation/ no scar on LGE-CMR

Beta-blocker therapy → effectively reduced subjective extrasystole burden

Return to training and competition → permitted under beta-blocker cover and symptom monitoring

PATHOPHYSIOLOGY OF *C. JEJUNI* CARDIAC INVOLVEMENT

C. jejuni gastrointestinal infection (contaminated water source)

Release of circulating toxins
→ direct mucosal damage + systemic spread

MOLECULAR MIMICRY
Homology between bacterial antigens and cardiac proteins (gangliosides)
→ cross-reactive immune response

Delayed immune-mediated myocardial inflammation (onset 3 weeks after clinical recovery)

Ventricular ectopy / arrhythmia
Exertional dyspnea, palpitations at rest

Resolution without structural damage (LGE-CMR negative at 3 months)

DISCUSSION

C. Jejuni and the heart :

Cardiac involvement: circulating toxins may cause acute myopericarditis.
Delayed immune-mediated myocardial injury occurs via molecular mimicry — bacterial antigens cross-reacting with host cardiac tissue

Delayed pattern : Symptoms emerged only 3 weeks after full clinical recovery from GI illness. This **strongly suggests an immunological rather than direct toxic mechanism**.

Cardiac MRI timing : CMR was performed >3 months after symptom onset — deliberate delay to allow oedema resolution and improve specificity for fibrosis/scar detection (LGE). Negative LGE-CMR reassuring but does not exclude an earlier inflammatory phase.

Why not asthma? The character of dyspnea — biphasic "chest obstruction", distinct from prior asthma flares — combined with temporal correlation with VES/NSVT on CPET recovery, redirected the diagnostic approach from pulmonology to sports cardiology.

CLINICAL TAKE-HOME MESSAGE

- Cardiac symptoms (chest pain, exertional dyspnea, palpitations) during or after gastrointestinal illness should raise suspicion for post-infectious cardiac involvement.
- In competitive athletes, a thorough cardiac evaluation — including CPET with full recovery phase monitoring — is required prior to return to play.
- Post-infectious VES/NSVT may not be captured by resting ECG or standard echocardiography: exercise-recovery monitoring is essential.
- Cardiac MRI should be performed >3 months after symptom onset to optimise sensitivity for myocardial scar (LGE) and reduce false-positive oedema signals.

CONCLUSION

Diagnosis

Exertional VES and NSVT during CPET recovery — reproducing symptoms — was the diagnostic key.
Post-infectious myocarditis should be considered when symptoms arise weeks after *C. jejuni* illness.

C. jejuni as cardiac trigger

Direct toxic and delayed immune-mediated (molecular mimicry) mechanisms are described. The temporal pattern here — symptoms 3 weeks post-recovery on return to training — points to an immunological process.

Targeted management

Low-dose beta-blockers effectively suppressed ventricular ectopy and permitted safe return to competition. LGE-negative CMR at 3 months provided reassurance and guided sport eligibility.

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