“ Reversible cerebral vasoconstriction syndrome : Not Just Another Bad Migraine ”

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BACKGROUND

- Reversible cerebral vasoconstriction syndrome (RCVS) is a clinical and radiological entity characterised by multifocal vasoconstriction of the cerebral arteries showing reversibility over a 12 week-period, associated with recurrent thunderclap headaches.
- Although mostly described in adults, it remains rare and possibly underdiagnosed in children and adolescents.
- We describe the case of a 12-year-old male patient presenting to the paediatric emergency department with atypical recurrent headaches.

PATIENT PRESENTATION

- 12-year-old boy with no previous relevant medical history, no recent trauma, no exposure to toxic substances or medications
- Severe thunderclap occipital headaches, lasting 30 min, recurring each evening for 2 days
- Associated with photophobia, vomiting during acute headaches episodes, autonomic symptoms (profuse sweating, elevated blood pressure (BP)
- Initial normal physical (incl. complete neurological) exam

TIMELINE, EVOLUTION AND INTERVENTIONS

7/10/2025

- Initial presentation
- Resolution of headaches after level 1 analgesia and IV hydration

→ **Discharged home**

10/10/2025

- Persistent pain with dysautonomia and elevated BP
- Normal ophtalmological exam
- Small vascular irregularities and doubt for subarachnoid hemorrhage (SAH) on MRI (*see below*), further excluded by CT-scan and lumbar puncture

→ **Suspicion of RCVS**
→ **Patient admitted**

13/10/2025

- Persistent elevated BP
- Acute event with right hemiparesis, paresthesia and dysarthria lasting for 10 min
- Multiple frontal acute ischemic lesions on repeat MRI *(see below)*
- Exacerbation of arterial stenosis on transcranial doppler ultrasond

→ **Introduction of nimodipine** *(see after)*

21/10/2025

- Headaches resolution
- Normalisation of BP
- Negative extensive aetiologic work-up

→ **Patient discharged home on nimodipine* treatment** (30 mg 2x/j = 1.2 mg/kg/j) = calcium channel blocker mainly used to prevent brain damage after a SAH

11-12/2025

- Near-resolution of the abnormalities previously seen on MRI
- Discontinuation of nimodipine

→ **Return to full sports activity**

RCVS IN FOCUS

Patho-physiology	Transient dysregulation of cerebral vascular tone secondary to sympathetic hyperactivity, endothelial dysfunction and oxidative stress. (1)
Triggers	Vasoactive drugs, systemic inflammatory, autoimmune diseases, genetic connective tissue or vascular development disorders, acute stress situations and cold bathes. (2)
Treatment	Rest, avoidance of vasoactive substances, BP control and pain management. (3) Calcium channel blockers, specifically nimodipine and verapamil, are frequently employed to reduce vasoconstriction. (4)
Complications	Ischemic strokes, intracerebral hemorrhage, SAH, posterior reversible encephalopathy syndrome (PRES) occuring within days to weeks after symptoms onset in one-third of cases due to vasoconstriction.
Prognosis	Favourable, with complete recovery even in the presence of ischemic lesions or associated PRES. Long-term recurrence remains poorly documented. (2)

KEY MESSAGES

- Consider RCVS in case of **sudden, intense and recurrent headaches**.
- **Repeat imaging** is often required, as the initial MRI may not show any abnormality.
- **Angio-MRI and transcranial doppler ultrasound** are indicated both for diagnosis and surveillance.
- **Supportive treatment** and **BP control** can prevent serious complications, such as ischemia.
- RCVS may be **idiopathic** but **triggering factors** should always be investigated.

IMAGING FINDINGS

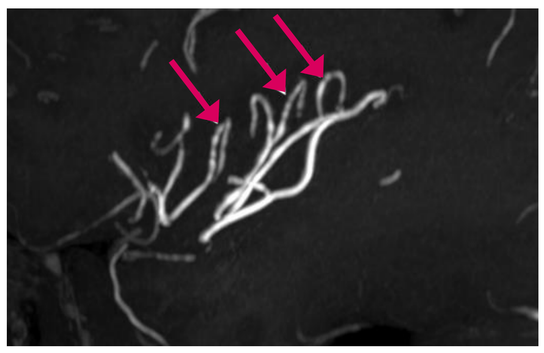

Fig 1. **String of beads** involving multiple branches of the right middle cerebral artery

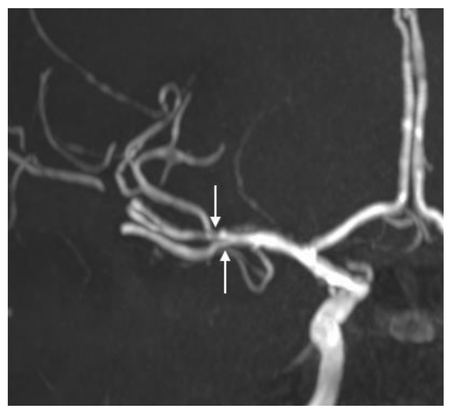

Fig 2. **Proximal narrowing** at M2 trifurcation of the right middle cerebral artery

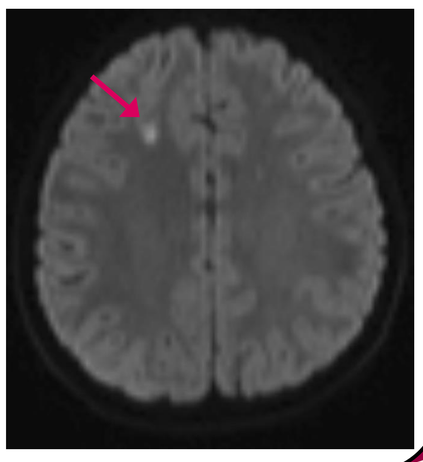

Fig 3. Watershed **acute ischemic focus** in the periventricular frontal white matter

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When the swim training turns toxic
chlorine gas exposure at a public swimming pool

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Introduction

Chlorine gas is a **highly reactive and toxic respiratory irritant** that poses significant health hazards through accidental or intentional exposure. Widely used as an industrial chemical and household disinfectant, chlorine intoxication can occur in various settings, including improper mixing of cleaning products or mishandling of swimming pool chemicals.

We report two cases of 15-year-old adolescents who developed **dyspnea and hypoxia** following accidental chlorine gas exposure caused by a **swimming pool pump malfunction** during a training session, resulting in gas release into the air. Both patients developed **severe symptoms after approximately 1–2 minutes** of exposure and were subsequently transferred to the pediatric emergency department (PED).

Case 1. The first patient had a known history of exercise-induced asthma. She immediately developed a cough, burning sensation of the upper airway. Received salbutamol and 100% O2 from prehospital providers. At the PED, she was HD stable but tachypneic with suprasternal and scalene retractions. Lung auscultation revealed decreased air entry on the right, diffuse bilateral crackles. Skin and eyes decontamination was performed. Oxygen therapy was continued with nebulized salbutamol/ipratropium bromide and IV methylprednisolone. Chest X-rays (Fig.1) demonstrated right upper lobe atelectasis with diffuse pulmonary infiltrates. Although an initial clinical improvement was observed, the patient rapidly deteriorated, prompting transfer to the intensive care unit for non-invasive ventilation (NIV). Bronchodilator therapy and systemic corticosteroids were continued for three days, and NIV was discontinued after four days. The clinical course was favorable, with planned pneumology follow-up.

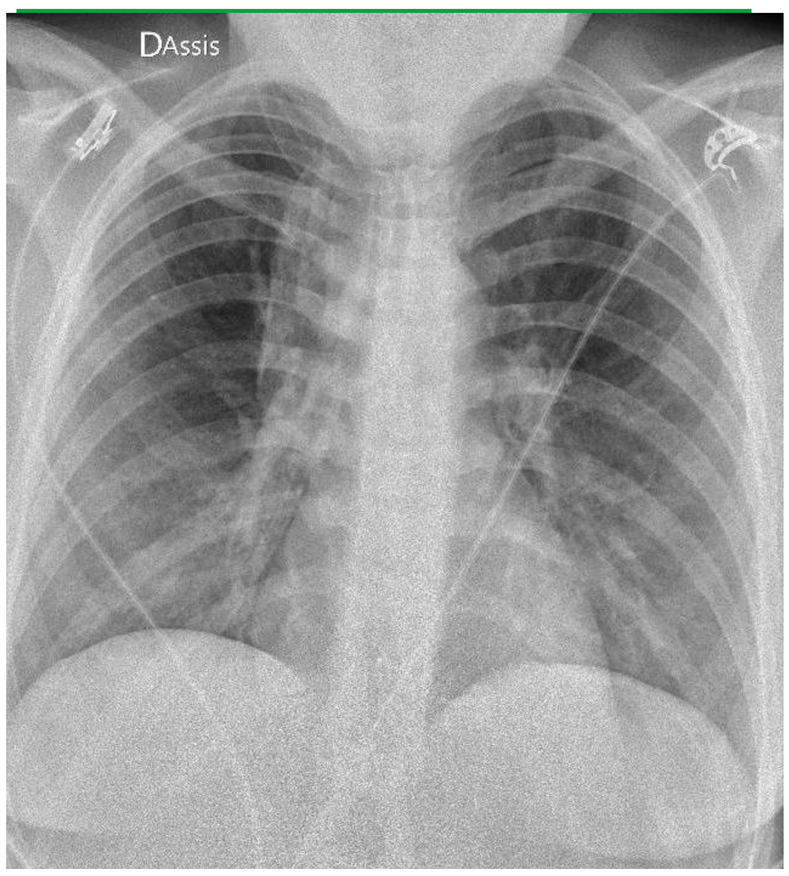

Fig.1: Chest XR of case 1. Right upper lobe atelectasis with diffuse pulmonary infiltrates.

Case 2. The second patient presented with an oxygen saturation of 91%, a respiratory rate of 42 breaths/min, dyspnea, and a sensation of throat discomfort. Management was similar to the first patient. Favorable clinical evolution and discharged after 24 hours of observation.

Chlorine gas inhalation

Acute Respiratory Effects. Chlorine inhalation induces **acute inflammation of the upper and lower airways.** Respiratory failure may occur immediately, subacutely, or in multiple stages.

Pathophysiology. The acid effect of chlorine and free radical oxygen species triggers a cascade leading to **alveolar edema, capillary leakage**, and ultimately **lung injury**.

Clinical Spectrum. Most patients experience immediate oropharyngeal irritation and cough, followed by:

- **Bronchospasm** and interstitial edema;
- Alveolar edema and capillary leakage;
- Acute lung injury and **ARDS**;
- Ophthalmic irritation requiring ocular irrigation.

Long-term Sequelae. Serious exposures may cause long-term pulmonary consequences such as **obstructive-type bronchial hyperreactivity** or **restrictive pulmonary disease.**

Take home message

Pool chemical risk ⚠ Chlorine vapors from swimming pool disinfectants represent a significant risk of **respiratory disorders** after inhalation. Even brief exposure (1–2 min) can lead to life-threatening respiratory failure.

Recognize, act fast and long-term follow-up ⚠ Treatment is supportive and long-term follow-up with objective pulmonary function testing may be necessary in serious chlorine exposure.

WHEN WORSENING IS NOT RELAPSE : CORTICOSTEROID WITHDRAWAL AFTER COMPLICATED OTOGENIC INFECTION

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BACKGROUND

Acute otitis media (AOM) complicated by mastoiditis may extend to :

- **Intracranial complications**
- **Cranial nerve deficits** (cases of petrositis)

Standard management :

- Intravenous (IV) broad-spectrum **antibiotics**
- **Surgical intervention**
- **Adjunctive corticosteroids (CS)** in selected cases to reduce inflammation

Early clinical deterioration after initial improvement raises concern for relapse or progression

- Represents a **diagnostic and therapeutic challenge**

Figure 3 — Timeline

DAY 0 : FIRST SYMPTOMS + INITIAL MANAGEMENT
Right mastoidectomy + tympanic paracentesis
IV Ceftriaxone
IV Metronidazole
IV Methylprednisolone

2 days later

DAY 2 : 48 HOURS AFTER INITIAL TREATMENT
Favorable clinical course
Stop Methylprednisolone
IV Ceftriaxone
IV Metronidazole

2 days later

DAY 4 : 48 HOURS AFTER CORTICOSTEROID WITHDRAWAL
Sudden secondary clinical deterioration

2 days later

DAY 6 : 48 HOURS AFTER CEFEPIME INTRODUCTION
Full recovery

CASE REPORT

CASE PRESENTATION (DAY 0)

16-year-old with complicated right AOM, with recent trip to Japan

- Mastoiditis + meningitis, cranial nerves VI–VII palsies, petrous apex abscess + epidural abscess

Management : IV ceftriaxone + metronidazole, mastoidectomy + paracentesis, IV CS (125 mg 1x/day = 2.3 mg/kg, during 48h)
Evolution : rapid clinical improvement

SECONDARY DETERIORATION (DAY 4)

Sudden clinical deterioration, headache, asthenia

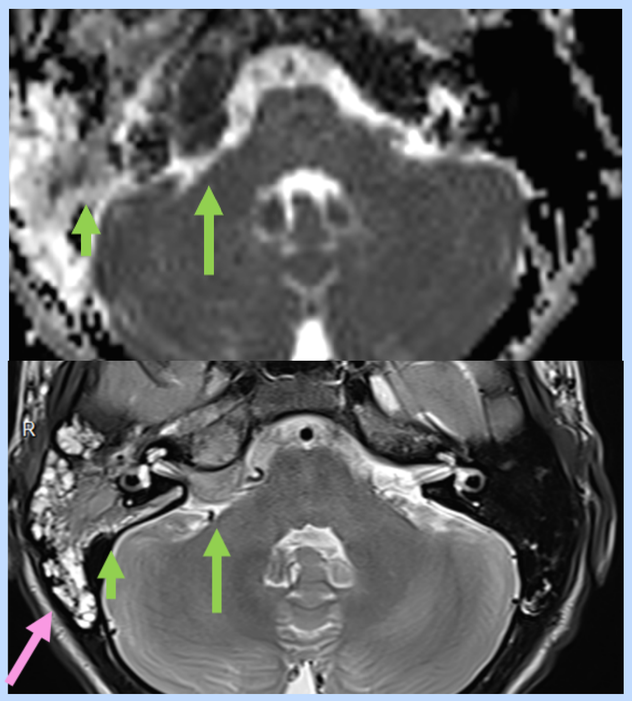
- Urgent MRI : new signs of meningitis, pyoventriculitis

Management : escalation to cefepime
Evolution : full recovery within 48h

OUTCOME & DIAGNOSIS

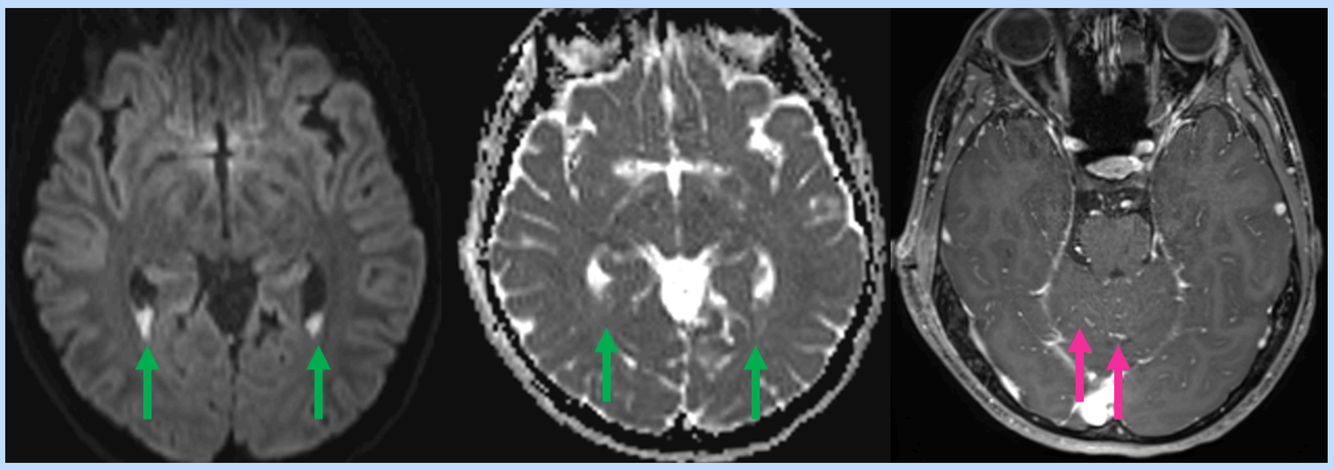
- PCR : Streptococcus intermedius → De-escalation to ceftriaxone
- MRI findings : compatible with delayed radiological evolution

Figure 1 — Brain MRI at Day 0



Secondary deterioration final diagnosis : CS withdrawal syndrome

Figure 2 — Brain MRI at Day 4



CONCLUSION

KEY MESSAGE

- Secondary neurological deterioration ≠ infectious relapse or progression
- **Consider CS withdrawal syndrome** when :
  - Symptoms recur after CS discontinuation
  - Inflammatory markers improving
  - Stable imaging

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Thoracic discomfort and fatigue in a young child: an atypical presentation of diabetic ketoacidosis

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Background

Diabetic ketoacidosis (DKA) remains a frequent and potentially life-threatening initial manifestation of diabetes mellitus in young children.
The nonspecific presentation of diabetic ketoacidosis frequently delays diagnosis, underscoring the importance of structured physician education to improve early symptom recognition and consideration of DKA in the differential diagnosis.

Case description

A 4-year-old boy presented to our Children's Permanence with thoracic discomfort and fatigue. Symptoms had progressed over a few days and were accompanied by reduced oral intake and mild pollakiuria. There was no history of fever, vomiting, or diarrhoea.

On our examination, the patient showed a reduced general condition, an increased work of breathing, while oxygen saturation remained normal. No abnormalities at the neurological examination were observed.

Laboratory investigations revealed severe hyperglycaemia (plasma glucose 20 mmol/L), metabolic acidosis (pH 7.28, bicarbonate 6.6 mmol/L, base excess −20 mmol/L), hyponatremia (Na+ 127 mmol/L), elevated anion gap (12 mmol/L) associated to a marked glucosuria and ketonuria, confirming the diagnosis of new-onset diabetes mellitus with DKA.

The patient was then admitted to hospital for metabolic stabilization and initiation of insulin therapy. Following clinical improvement, structured diabetes and nutritional education was provided to the parents.

Although initially challenging due to the patient's young age, repeated counselling and initiation of continuous glucose monitoring improved caregiver understanding.

At discharge, the patient was in good general condition, and showed no abnormal findings on physical examination.

Key symptoms

- Thoracic discomfort
- Fatigue
- Mild pollakiuria

Key laboratory results

Plasma glucose	20 mmol/L
pH	7.28
Bicarbonates	6.6 mmol/L
Urine glucose	500 mmol/L
Ketones	80 mmol/L

Conclusion

This case underscores the importance of considering diabetes mellitus and DKA in young children presenting with nonspecific symptoms such as fatigue and thoracic discomfort.
Multidisciplinary inpatient management, early caregiver education, and the use of modern glucose monitoring technologies are key to achieving safe transition to outpatient care in newly diagnosed paediatric diabetes.

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MOG antibody-associated encephalitis induced by Mycoplasma pneumoniae infection : case report.

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Background

Myelin oligodendrocyte glycoprotein antibody–associated disease (MOGAD) is an inflammatory demyelinating disorder of the central nervous system caused by IgG antibodies against MOG. Unlike acute disseminated encephalomyelitis (ADEM), MOGAD is associated with a higher risk of relapse. Neurological complications linked
to Mycoplasma pneumoniae infection with anti-MOG antibodies have recently been described. Here we present a new case.

Case presentation

We report the case of a 12-year-old boy admitted with a two-week history of fever, cough, and headache. Investigations revealed moderate inflammatory markers, left basal pneumonia on chest X-ray, and a positive nasal PCR for Mycoplasma pneumoniae. Despite azithromycin treatment, the patient developed neurological deterioration including altered consciousness (GCS 10/15), ataxia, urinary retention, hypotonia, and lethargy. Brain MRI showed lesions consistent with acute disseminated encephalomyelitis, with rapid radiological progression and seizures. High titers of anti-MOG antibodies were detected. The patient required pediatric intensive care, mechanical ventilation, and was treated with intravenous immunoglobulins, high-dose corticosteroids, plasmapheresis, and tocilizumab. He developed tetraparesis and dysarthria, requiring prolonged rehabilitation, with gradual recovery and independent ambulation after three months.

Discussion

Only a few cases of MOG-associated encephalitis triggered by Mycoplasma pneumoniae have been reported. Proposed mechanisms include cytokine-mediated inflammation, immune cross-reactivity between mycoplasma antigens and myelin, and inflammatory vascular involvement. Diagnosis relies on evidence of Mycoplasma pneumoniae infection, early detection of MOG-IgG antibodies, and characteristic MRI findings. Treatment includes corticosteroids, intravenous immunoglobulins, and macrolides. Although prognosis is generally favorable, long term follow-up is essential due to relapse risk.

Conclusion

Mycoplasma pneumoniae, though primarily a respiratory pathogen, can cause severe neurological complications. It should be as a differential diagnoses in patients presenting with encephalitis or unexplained neurological symptoms.

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Myelin oligodentrocyte glycoprotein (MOG) antibody-associated encephalitis induced by Mycoplasma pneumoniae infections, Yan-ru Liu, Xiang-Dong Zheng, Ying Xiong, septembre 2024. DOI : https://doi.org/10.1186/s13052-024-01768-w

Multifocal osteomyelitis in a child with sickle cell disease

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Background

Children with **sickle cell disease** (SCD) are at **increased risk** of **severe bacterial infections** and **osteoarticular complications**. **Differentiating bone infarction** from **osteomyelitis** remains **challenging** because both conditions may present with fever, bone pain and non-specific imaging findings.

Case Report

11-year-old boy with **homozygous sickle cell** disease and a history of **recurrent vasoocclusive crises** (VOC)

Clinical presentation :

- **Progressive bone pain** in the right humerus and left knee
- **Four-week** history of **intermittent fever**
- Prior intravenous antibiotic courses in Nigeria

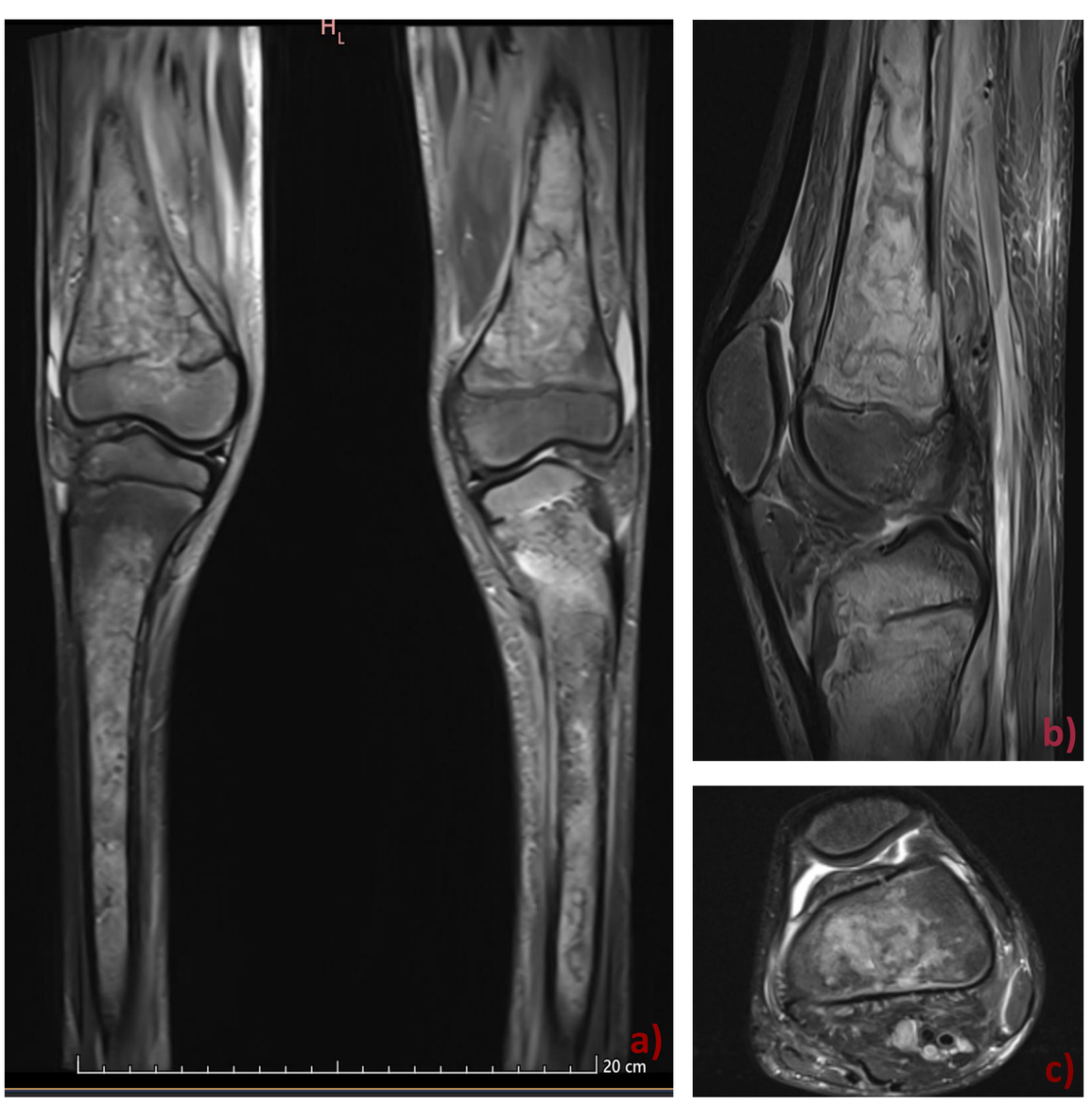
On admission :

- **Septic** appearance, hypotensive (84/49 mmHg) and tachycardic (138 bpm)
- Initially **febrile** at 40.3°C, then **hypothermic** (34.9°C)
- **Swelling, erythema and tenderness** of the right elbow and left knee joint
- Lab workup : **severe anemia** (40 g/l) and **markedly elevated inflammatory markers** (CRP 153 mg/l)
- No evidence of septic emboli or endocarditis on brain MRI, fundoscopic examination and echocardiography

Microbiological findings :

- **Bone biopsies** : ESBL-producing ***Klebsiella pneumoniae*** and ***Enterobacter cloacae***
- Known **digestive colonisation** with ESBL-producing ***Klebsiella pneumoniae***
- Negative blood cultures

! Prior hospitalisations and antibiotic exposure likely contributed to multidrug-resistant bacteria selection



Radiological findings

Whole body MRI : multiple osteomyelitis foci associated with extensive bone infarctions of the long bones and several thoracic vertebrae
STIR (a and b) and T2 Fat Sat (c) of distal femur and proximal tibia :

- Extensive **bone marrow signal abnormalities**
- **Periosteal** reaction
- **Diffusion restriction**
- Adjacent **soft-tissue infiltration**

Treatment

Meropenem + vancomycin
↓
Amikacin added
↓
Vancomycin stopped with reception of bone biopsies
↓
Persistent infection
Surgical debridement of necrotic bone tissue
↓
3 months continuous **meropenem** infusion

Catamnesis

Fever resolved 19 days after surgery, alongside gradual **clinical** and **biological improvement** (decreasing inflammatory markers, resolution of symptoms and functional recovery)

Take-home messages

✓ Diagnostic of osteomyelitis in SCD remains challenging as **clinical features** may **overlap** with **VOC**
✓ **Peristent fever** and **multifocal bone pain** should raise **suspicion** for **osteomyelitis**
✓ **Gram-negative pathogens** must be **covered** empirically because of possible **intestinal bacterial translocation** in SCD patients
✓ Surgical source control may be necessary for infection resolution
✓ Early **imaging**, **microbial sampling** and **multidisciplinary** management are essentials

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