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RCP Examiner
RT 4 Tanzania
30 October


Ça va Cameroon

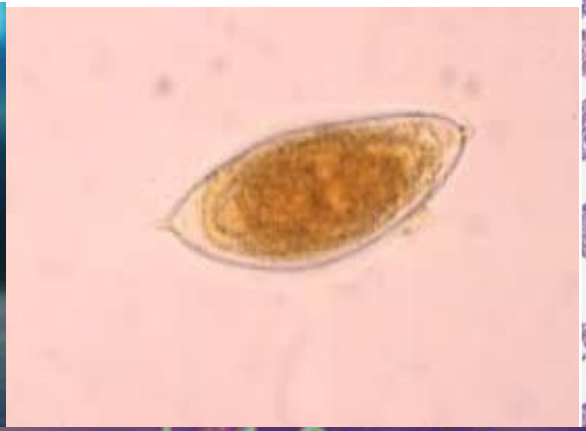

Doc,
I can't feel
my legs!!
?!!?!?

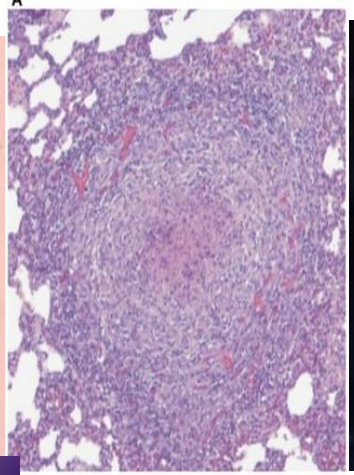
Gathering my thoughts together

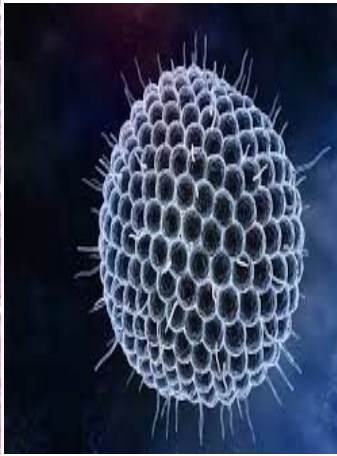
- Myelopathy
  - osteomyelitis
  - Discitis
  - Epidural abscess
  - granuloma
- Myelitis
  - MS
  - NMO
  - Post vaccine
  - Auto antibodies
  - Cancer
  - virus
- Vascular
  - Hemorrhage AVM angioma
  - Ischemia Artery vein

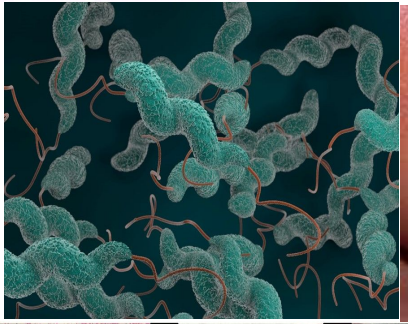
- Motor weakness
- Stiffness
- Spasm
- Sensory symptoms
- Deep root pain
- Sphincteric disturbance
- Anxiety
- Depression
- Death

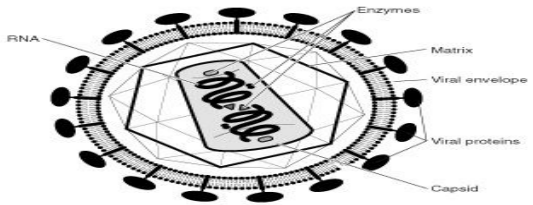
- UMN
- LMN



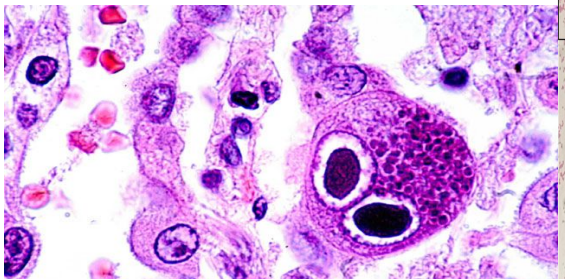


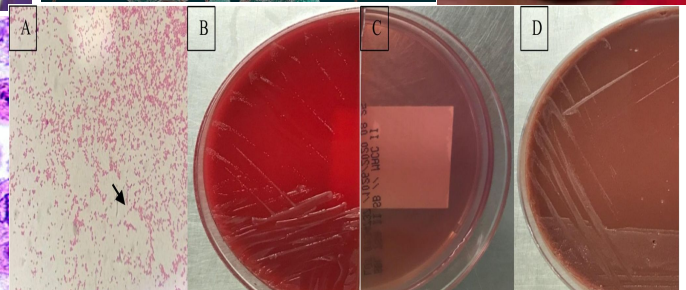






Structure of Human Immunodeficiency Virus (HIV)

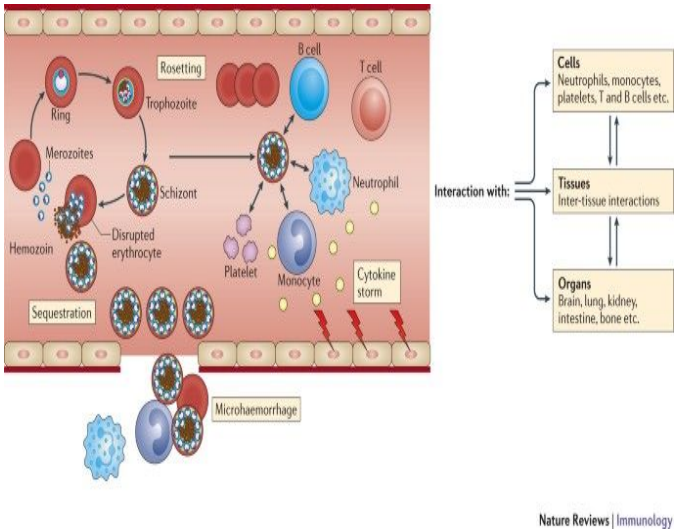




Pathophysiology of infection in the spinal cord

- Humoural Immunity: antigen antibody driven by b cells
- T-cells macrophages cytokines driven by antigens
- Genetics: caucasian and black
- Antibody storm
- Cytokine storm
- Vasculitis
- Demyelination
- Edema, necrosis and scarring
- Steroids have no role in malaria

Pathophysiology example



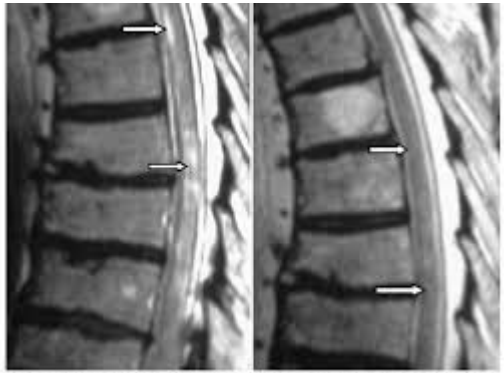
Risk factors

- IV drug abuse
- HIV
- Long term steroids
- Diabetes Mellitus
- Organ Transplant
- Malnutrition
- Cancer
- Surgery
    - Long duration
    - Blood loss
    - Implantation of instruments

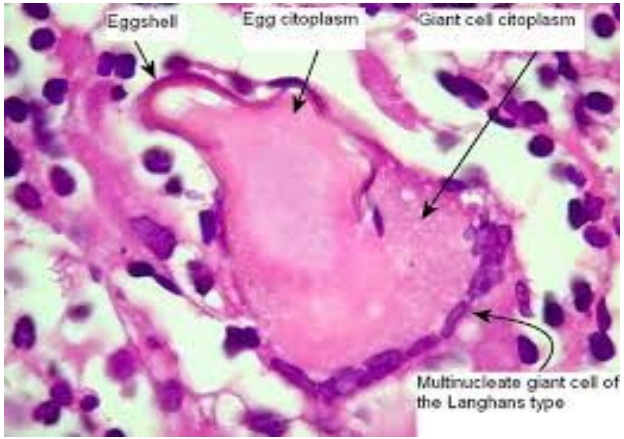
Root of infection

- Direct
- Blood
- surgery

Schistosomiasis



Sagittal T1-weighted MR images of the thoracic spine (left) before treatment (arrows) continue to diffuse into



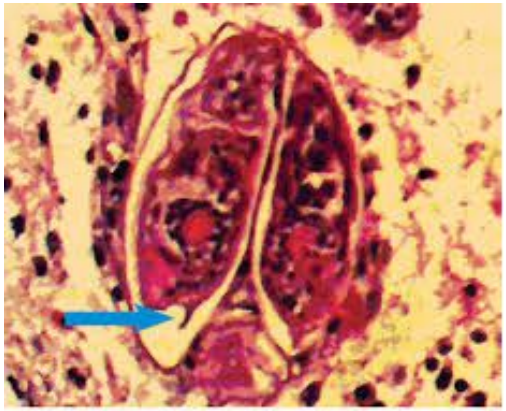
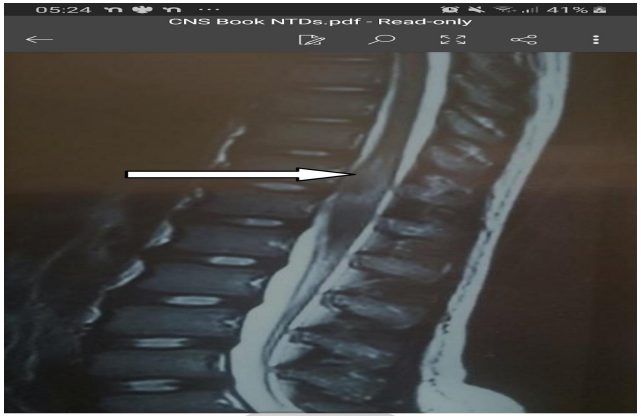
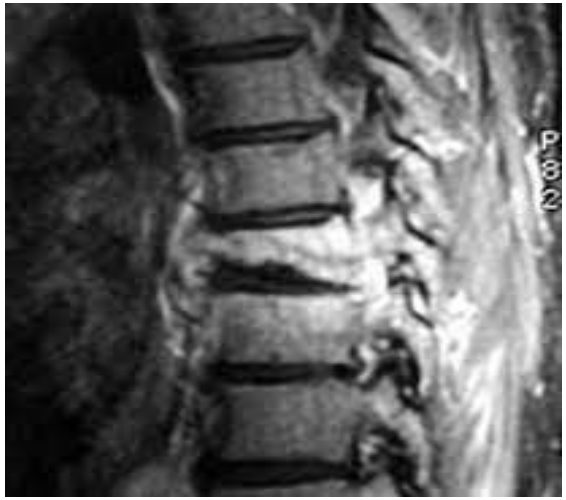


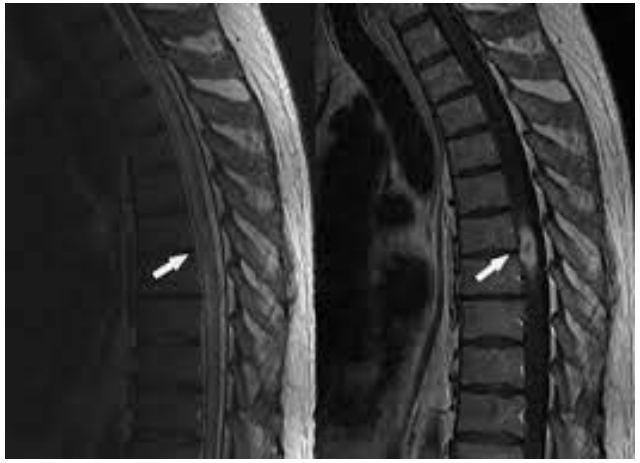
Figure 1 Two schistosomal eggs are seen: the one on the left has a lateral spine

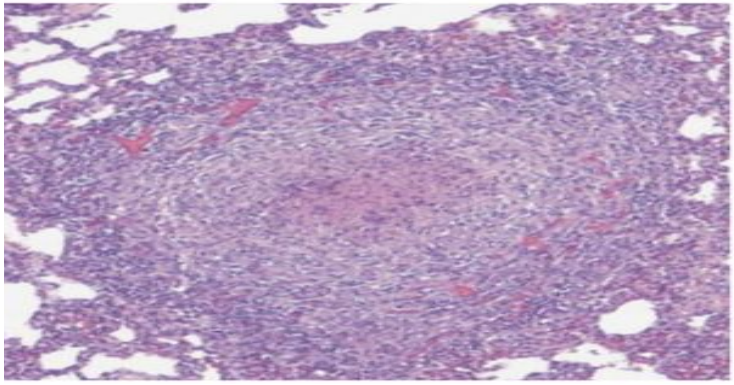


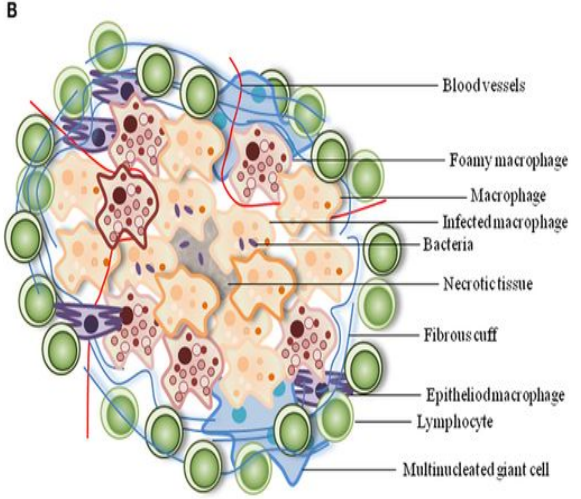
schistosomiasis presented as tr

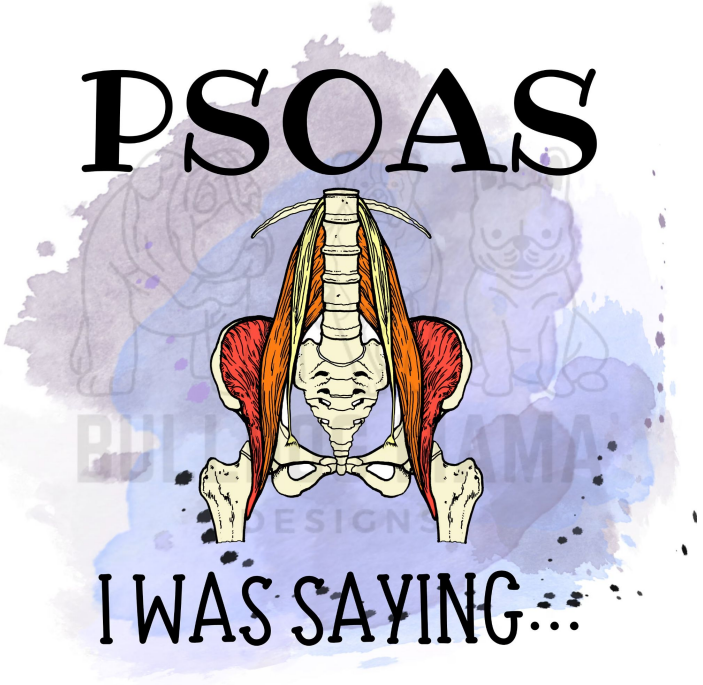
Tuberculosis

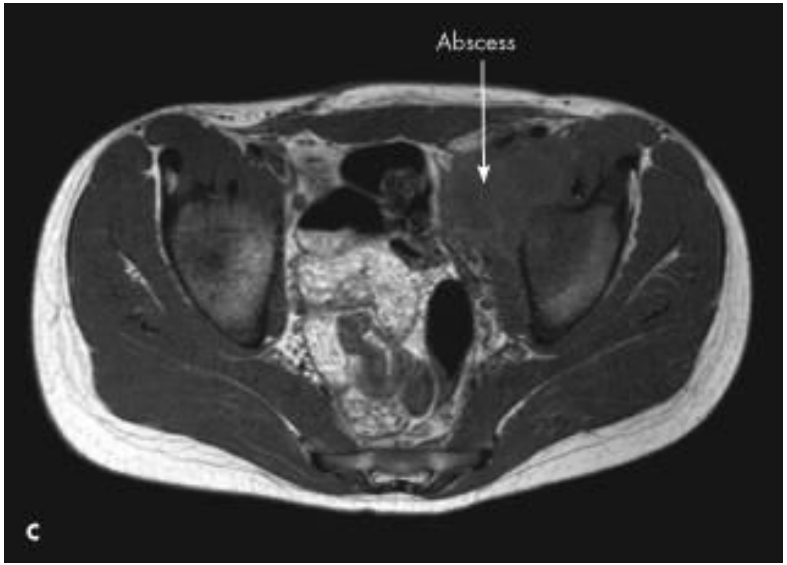




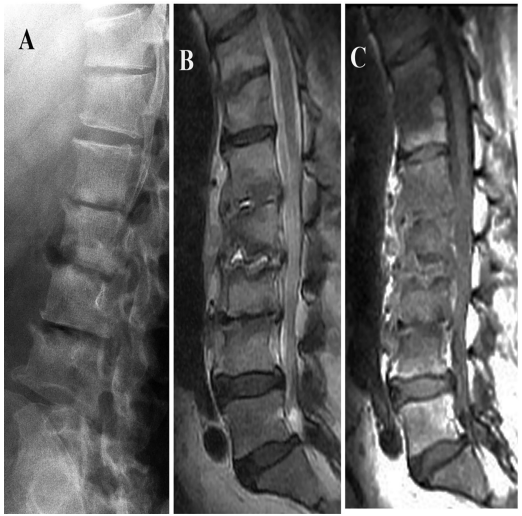


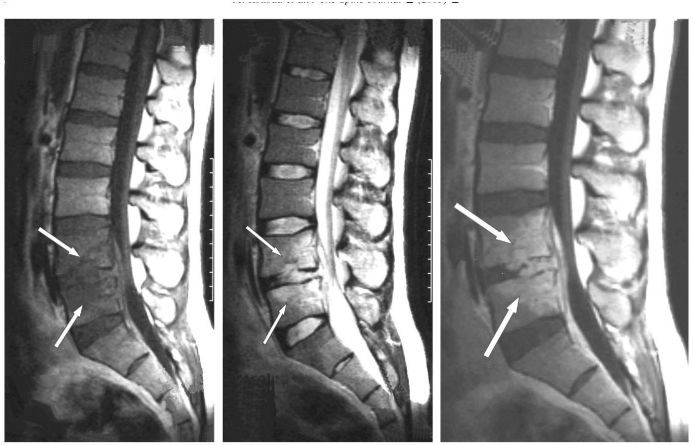


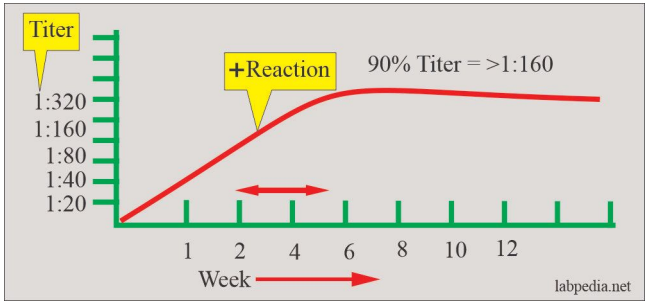

PSOAS
I WAS SAYING...


Abscess
C

Brucellosis

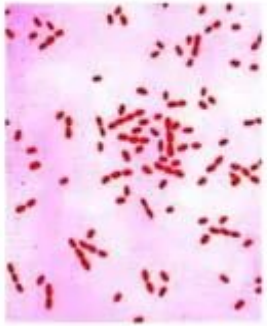




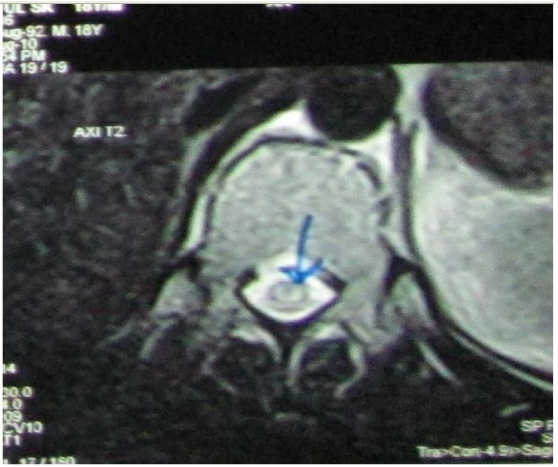


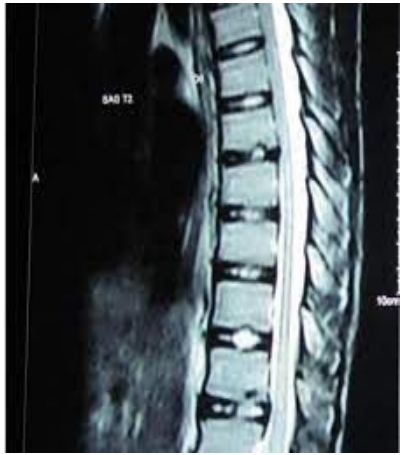
MORPHOLOGY

- Brucellae species are small, gram-negative aerobic coccobacilli, 0.5-0.7 µm x 0.6-1.5 µm in size.
- They are nonmotile, noncapsulated, nonsporing and non-acid fast.



Malaria species





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Delayed cerebellar ataxia induced by *Plasmodium falciparum* malaria: A rare complication

Emmanuel Edwar Siddig, Sarah Misbah El-Sadig, Hala Fathi Eltigani, Ahmed Mudawi Musa, Nouh Saad Mohamed, Ayman Ahmed

First published: 20 October 2023 | https://doi.org/10.1002/ccr3.8053

22:29 4G 82

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vivax in the pathogenesis of severe malaria which is characterized by the features of different organ dysfunctions, which were previously thought to be caused by P. falciparum alone. Though several case studies have mentioned the association of the P. vivax infection with cerebral malaria, a causal correlation has yet to be established. Dorsal cord myelitis (which leads to paraplegia) during the febrile illness, is rarely described in association with vivax malaria, though there are reports on the Post Malaria Neurological Syndrome (PMNS) and acute disseminated encephalomyelitis following vivax malaria. We are reporting a case of P. Vivax malaria which presented with myelitis, which responded well to the antimalarial treatment.

Keywords: Neurological complication, P. vivax malaria, Dorsal cord myelitis

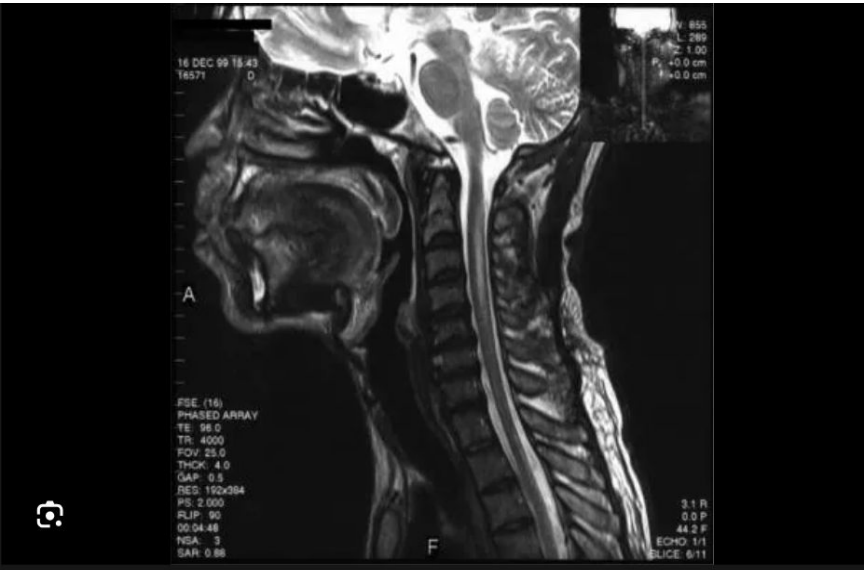
Feedback

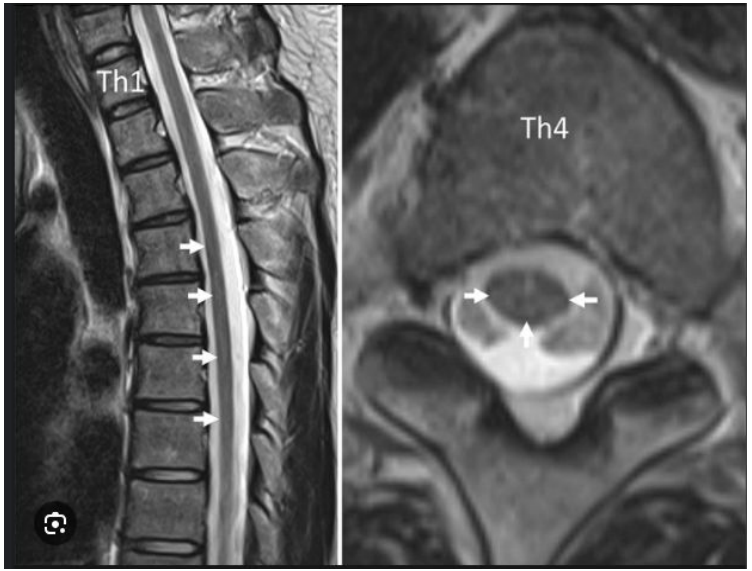
INTRODUCTION

AA ncbi.nlm.nih.gov

HIV Associated Vacuolar Myelopathy

Human immunodeficiency virus-associated vacuolar encephalomyelopathy





Fungal infection

Current Medical Mycology

2020, 6(1): 55-60

The first rare and fatal case of invasive aspergillosis of spinal cord due to *Aspergillus nidulans* in an Iranian child with chronic granulomatosis disease: review of literature

Mahin Tavakoli[1], Mohammad Taghi Hedayati[2], Hossein Mirhendi[3], Sadegh Nouripour-Sisakht[4], Newsha Hedayati[5], Fatemeh Saghafi[6], Setareh Mamishi[7*]

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Email: Smamishi@gmail.com

A B S T R A C T

Background and Purpose: Invasive aspergillosis (IA) of the central nervous system (CNS) is a devastating complication which is rarely reported in immunocompromised children.
In this case presentation, we reported a rare and fatal IA with spinal cord involvement in a 10-year-old child with X-linked chronic granulomatosis disease (CGD).
Case report: The child had a previous history of pulmonary tuberculosis. A cervical spine X-ray revealed the involvement of cervical vertebrae (T4/T5) and ribs causing spinal cord compression and epidural abscess. The patient underwent a decompressive laminectomy and mass removal. The histopathology and culture results suggested IA. Despite the aggressive and prolonged therapy, he died within one year. *Aspergillus nidulans* was identified as the causative agent based on morphological and molecular studies.
Conclusion: This synopsis represents the aggressive behavior of infection caused by *A. nidulans* in the CGD patient.

Keywords: *Aspergillus nidulans*, Chronic granulomatosis disease, Invasive aspergillosis, Spinal cord

➢ **How to cite this paper**
Tavakoli M, Hedayati MT, Mirhendi H, Nouripour-Sisakht S, Hedayati N, Saghafi F, Mamishi S. The first rare and fatal case of invasive aspergillosis of spinal cord due to *Aspergillus nidulans* in an Iranian child with chronic granulomatosis disease: review of literature. Curr Med Mycol. 2020; 6(1): 55-60. DOI: 10.18502/cmm.6.1.2551

Introduction

Chronic granulomatous disease (CGD) is a rare inherited disorder of phagocytic cells caused by defects in the nicotinamide adenine dinucleotide phosphate oxidase complex [1]. It may be diagnosed in childhood or adulthood; however, the majority of the affected patients are children under five years of age [2]. These patients may present with few to mild nonspecific clinical symptoms without fever or leukocytosis, even when seriously infected [1].

Although high levels of erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP) may be the only indicators, serum CRP is more useful than ESR for the diagnosis and monitoring of infection in CGD patients [3]. The CGD patients may have concurrent bacterial and fungal infections [4]. In an attempt to identify patients with documented bacterial or fungal infections, the medical records of 268 patients with CGD were followed at a single center over 4 decades [4]. In the mentioned study, the incidence of fungal infections was restricted to *Aspergillus* species, with *Aspergillus fumigatus*, followed by *A. nidulans*, accounting for a higher proportion of IA [4].

Aspergillus nidulans is one of the most important and well-known species of the *Aspergillus* section *Nidulantes* [5]. Although *A. fumigatus* has been by far reported as the most common pathogen, *A. nidulans* is reportedly the most virulent pathogen disseminating to the adjacent bones and then to the brain, thereby resulting in mortality [4]. To date, diverse clinical cases caused by *A. nidulans* have been reported

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- Rheumatoid arthritis Biological treatment:IGRA positive TB
- 48 year old Farmer with lower limb weakness and high blood pressure
- Bus driver lower limb weakness and pain on lying on his back
- 58 year old university lecturer following a GIT surgery 6 weeks later lower limb weakness
- 42 year old IT specialist after a visit to Thailand lower limb weakness intubated

14:08 4G 50

A 14-Year-Old Boy from Rural Tanzania With Difficulty in Walking

William P. Howlett, in
Clinical Cases in Tropical Medicine (Second Edition), 2022

Answer to Question 1

What is the Clinical Diagnosis?

The clinical syndrome is spastic paraparesis. The main differential diagnosis in Africa includes spinal tuberculosis (Pott's disease), transverse myelitis, spinal cord infections such as schistosomiasis and tuberculous myelitis, spinal malignancy (mainly metastases) and tropical nutritional myeloneuropathies.

There are three important features in our case: (1) the isolated involvement of motor neurons without any sensory and bladder involvement; (2) the absence of back pain; and (3) the acute onset with no progression over 2 years. These three clinical points make spinal tuberculosis, spinal cord infection or spinal malignancy very unlikely. Of note, his diet (and probably that of his siblings and other children in the village) for the 2 months before the illness was almost exclusively cassava, and the sa

FEEDBACK

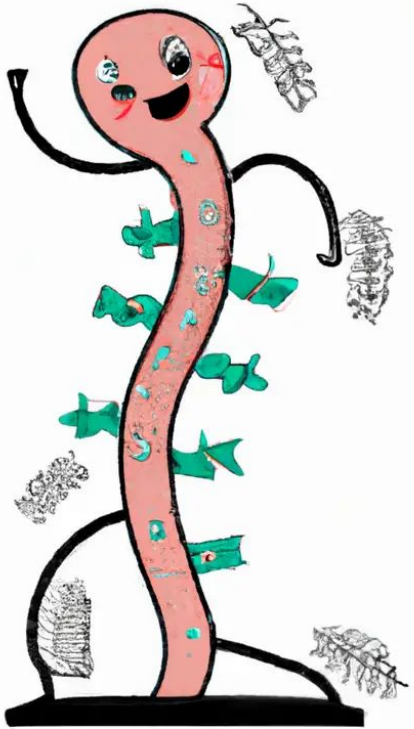
sciencedirect.com

14:08 4G 49

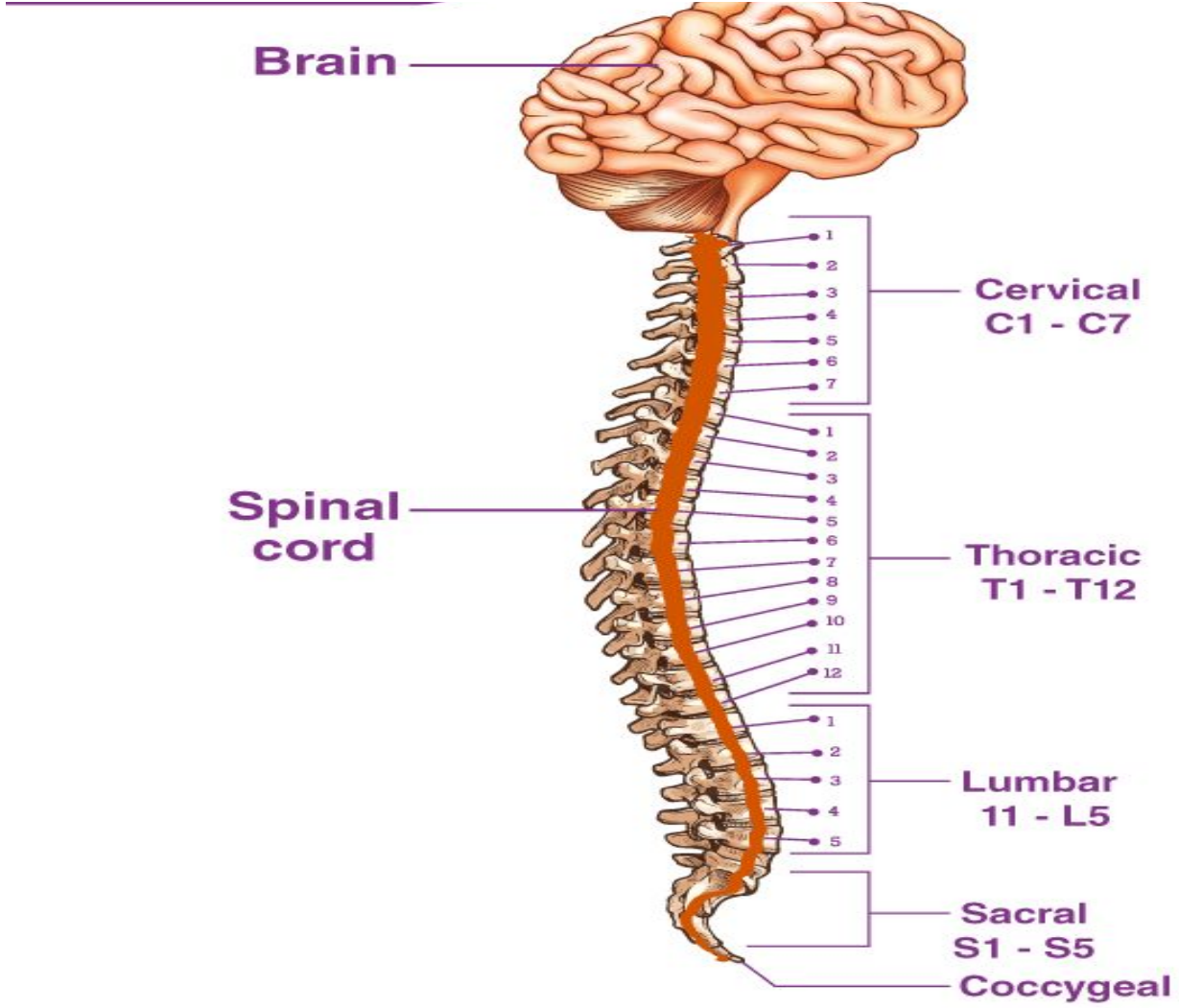
exclusively cassava, and the same disease has affected one of his siblings and more children in the neighborhood. Hence, a nutritional cause must be suspected.

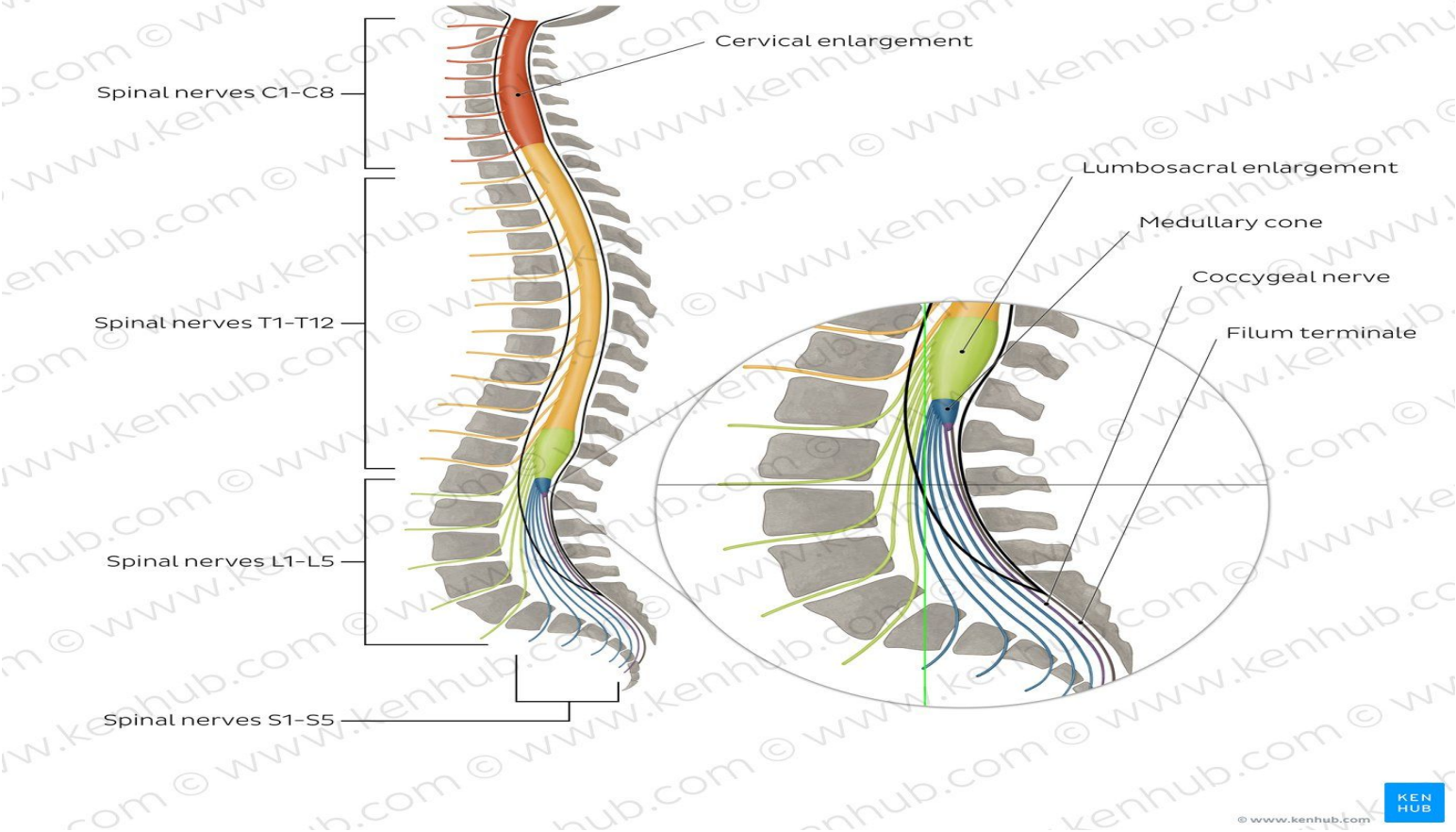
The tropical myeloneuropathies that are nutritional in origin are konzo and lathyrism. Lathyrism in Africa occurs exclusively in Ethiopia. The clinical diagnosis in our patient is konzo. Konzo is a distinct form of tropical spastic paraparesis which occurs exclusively in cassava-growing areas in Africa.

• **Fig. 24.1** A 14-year-old boy from rural Tanzania with spastic paraparesis. His illness started about 2 years earlier and had an acute onset. Several other people are also affected in his own and neighbouring villages.



Spinr


Brain
Spinal cord
1
2
3
4
5
6
7
Cervical
C1 - C7
1
2
3
4
5
6
7
8
9
10
11
12
Thoracic
T1 - T12
1
2
3
4
5
Lumbar
11 - L5
Sacral
S1 - S5
Coccygeal



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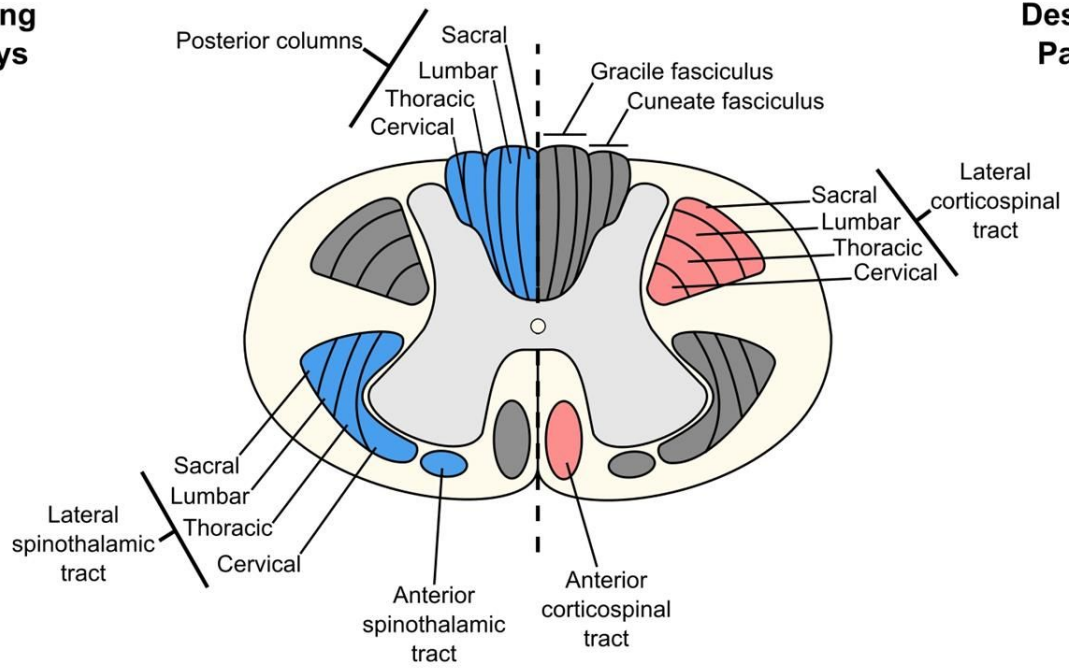
Spinal Cord Pathways

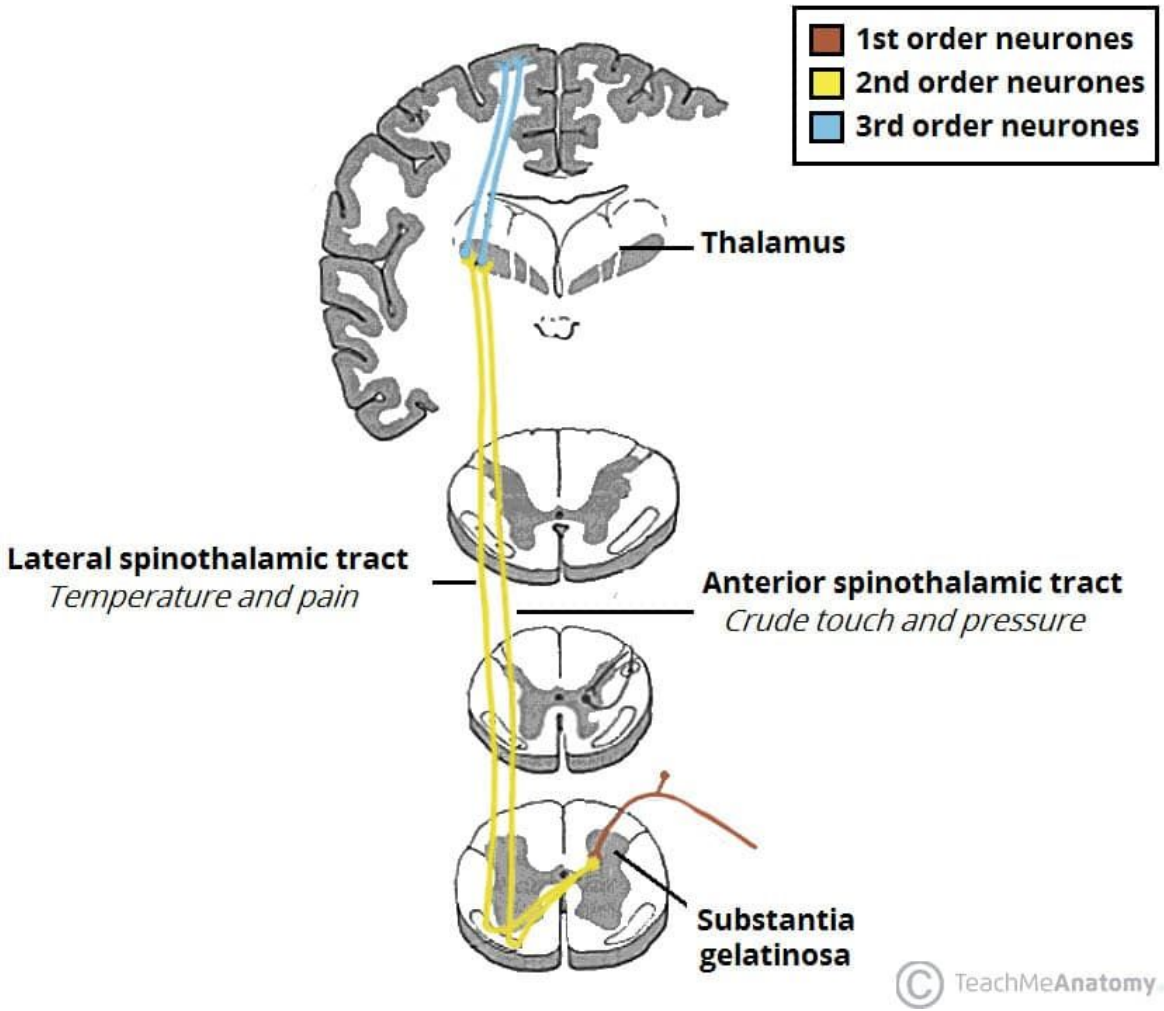
Ascending Pathways

Descending Pathways



© Lineage

Moises Dominguez

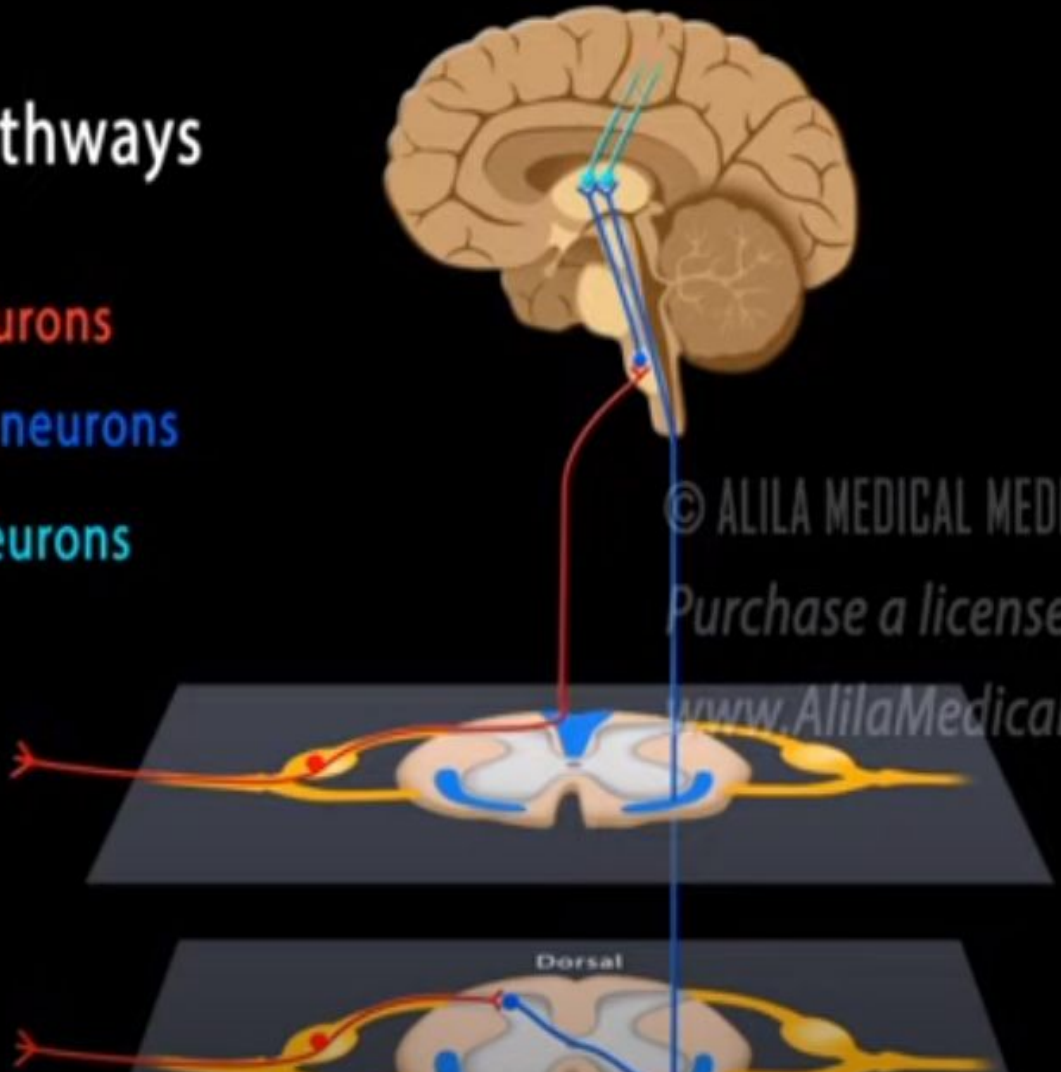

1st order neurones
2nd order neurones
3rd order neurones
Thalamus
Lateral spinothalamic tract
Temperature and pain
Anterior spinothalamic tract
Crude touch and pressure
Substantia gelatinosa
© TeachMeAnatomy

Sensory pathways

First-order neurons

Second-order neurons

Third-order neurons



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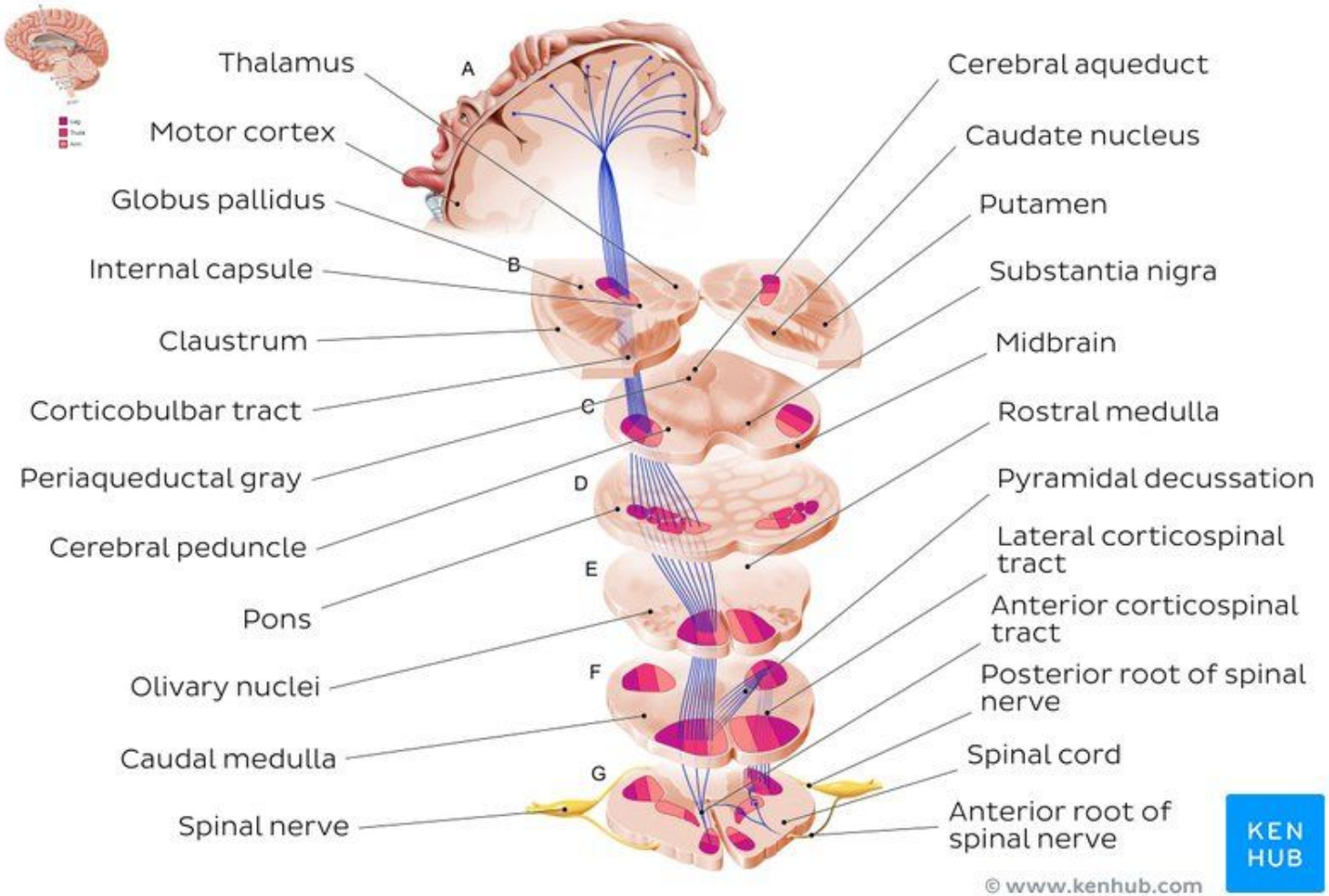
Thalamus
Motor cortex
Globus pallidus
Internal capsule
Claustrum
Corticobulbar tract
Periaqueductal gray
Cerebral peduncle
Pons
Olivary nuclei
Caudal medulla
Spinal nerve
A
B
C
D
E
F
G
Cerebral aqueduct
Caudate nucleus
Putamen
Substantia nigra
Midbrain
Rostral medulla
Pyramidal decussation
Lateral corticospinal tract
Anterior corticospinal tract
Posterior root of spinal nerve
Spinal cord
Anterior root of spinal nerve
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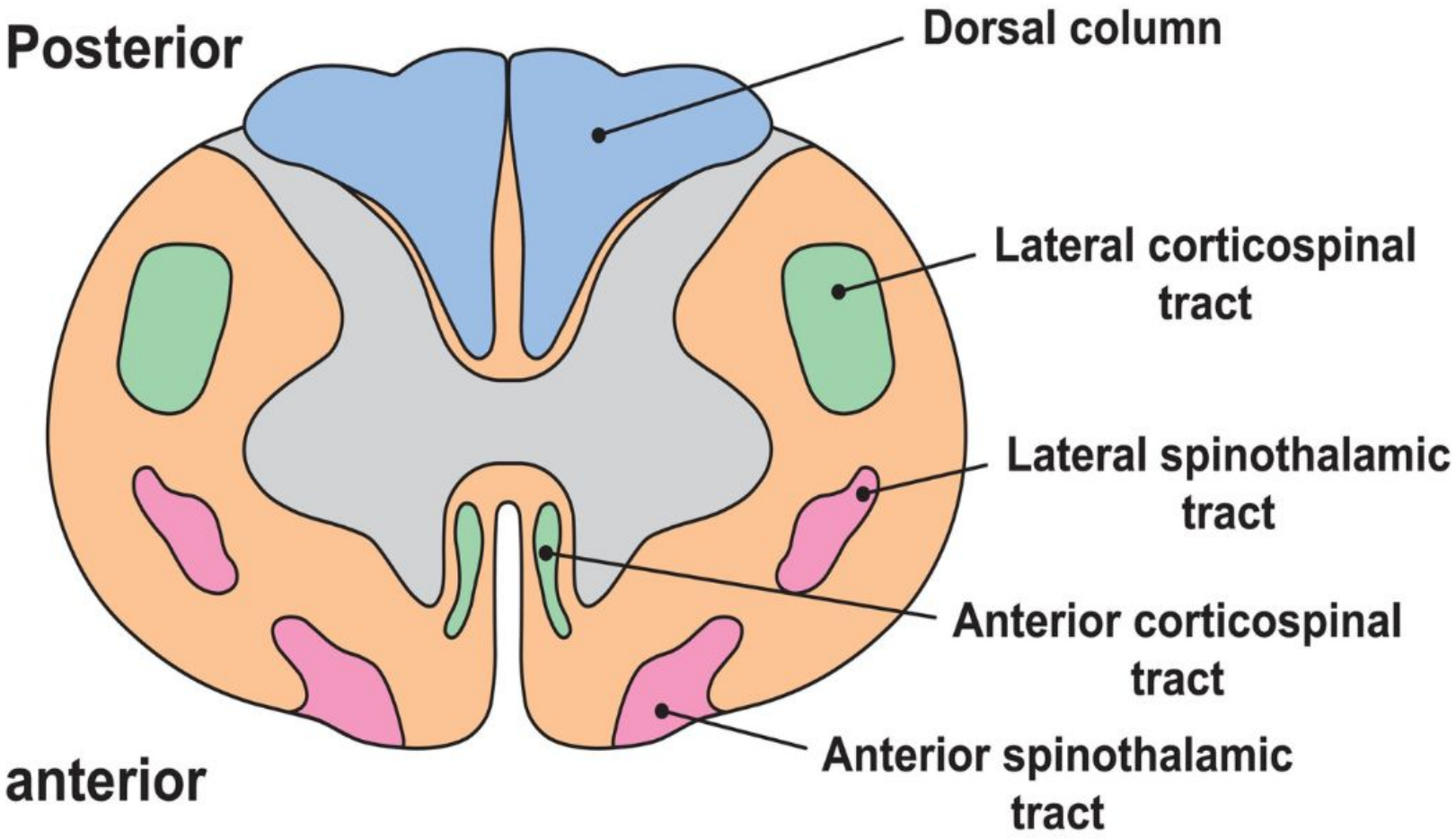

Posterior
Dorsal column
Lateral corticospinal tract
Lateral spinothalamic tract
Anterior corticospinal tract
Anterior spinothalamic tract
anterior

Descending Tracts (Motor)

Lateral Corticospinal Tract
- main voluntary motor
- upper extremity motor pathways are more medial (central)

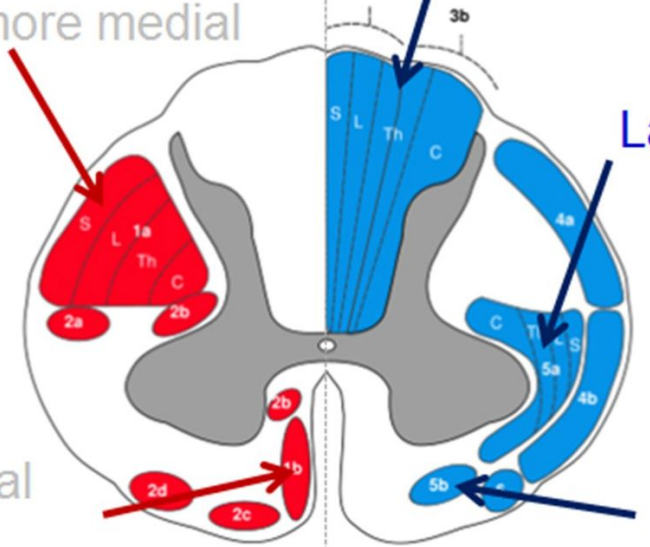
Ventral Corticospinal Tract
- voluntary motor

Ascending Tracts (Sensory)

Dorsal Columns (posterior funiculi)
- deep touch, proprioception, vibratory

Lateral spinothalamic tract
- pain and temperature

Ventral spinothalamic tract
- light touch



DESCENDING SPINAL CORD PATH

- **UPPER MOTOR NEURONS:** transmit MOTOR COMMANDS from BR
- **LOWER MOTOR NEURONS:** INNERVATE MUSCLES

CORTICOSPINAL TRACT

~ CONTROLS VOLUNTARY MOVEMENT of MUSCLES

ANTERIOR CORTICOSPINAL TRACT

└ MUSCLES of the TRUNK

LATERAL CORTICOSPINAL TRACT

└ MUSCLES of the LIMBS

RUBROSPINAL TRACT

~ ORIGINATES in RED NUCLEUS

RETICULOSPINAL TRACT

~ ORIGINATES in RETICULAR FORMATION

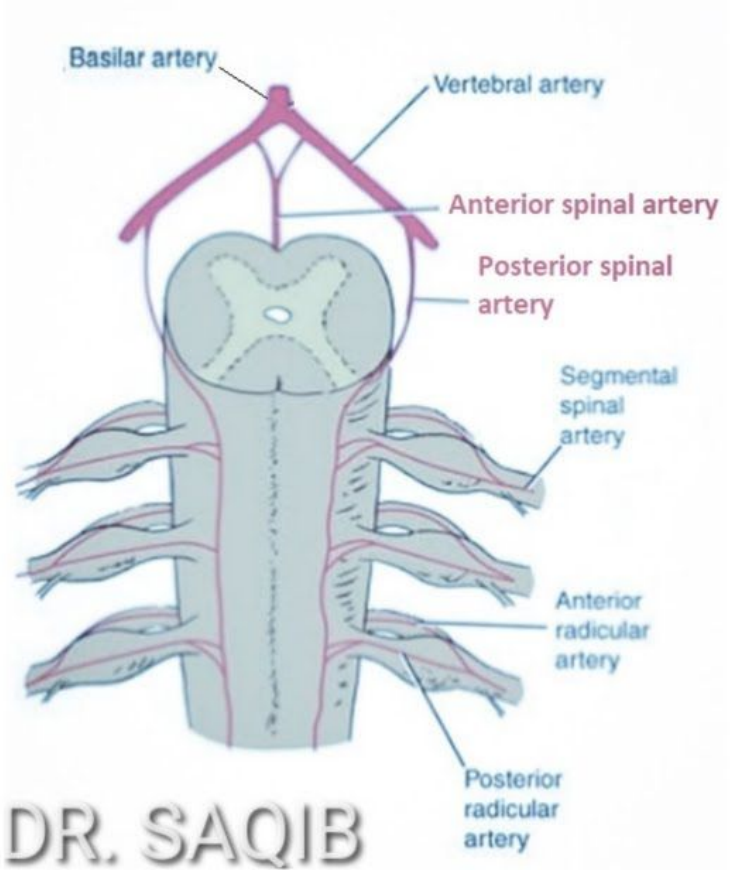
TECTOSPINAL TRACT

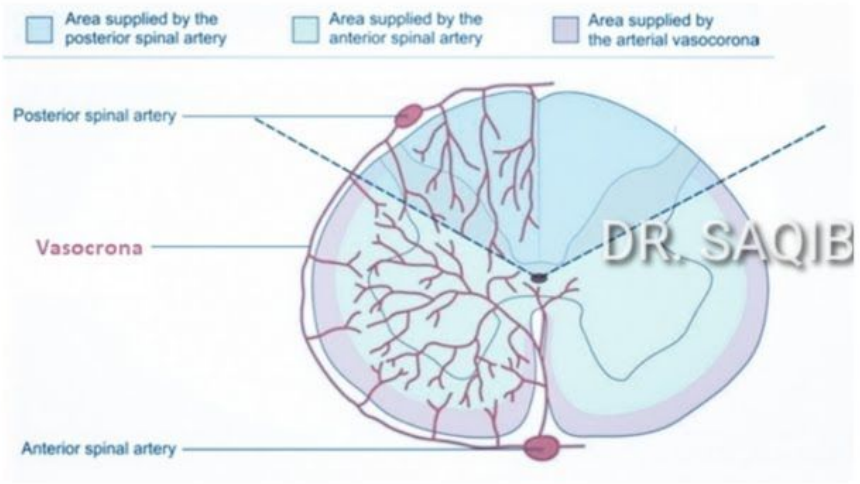
~ ORIGINATES from DORSAL MIDBRAIN

VESTIBULOSPINAL TRACT

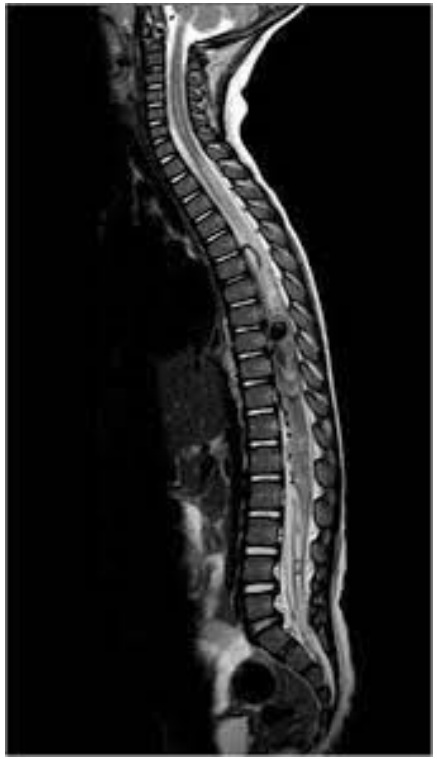
~ ORIGINATES from the VESTIBULAR NUCLEI

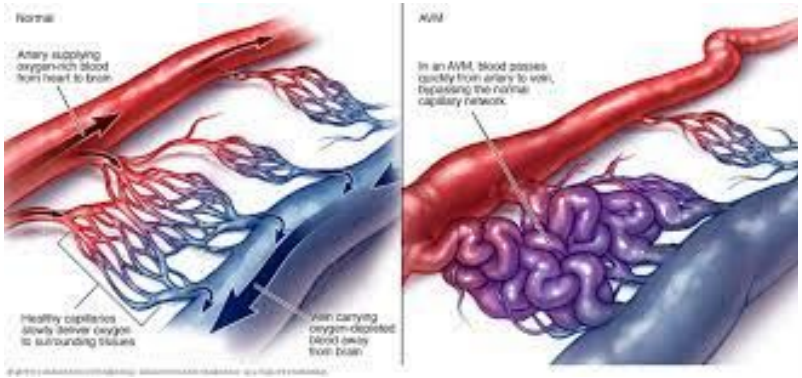
BLOOD SUPPLY OF SPINAL CORD

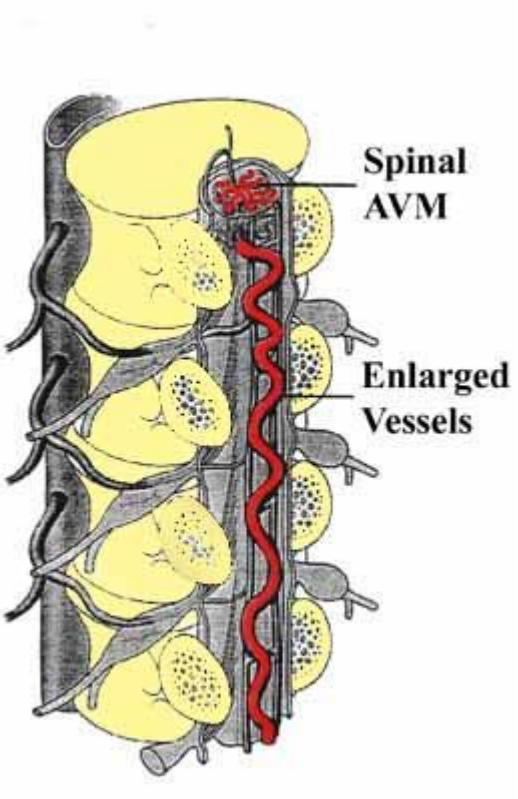




Arteriovenous malformation

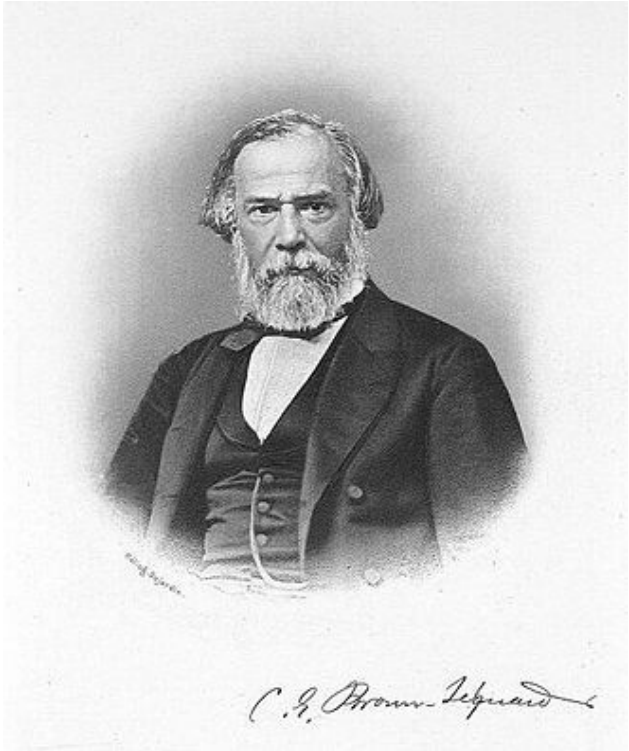


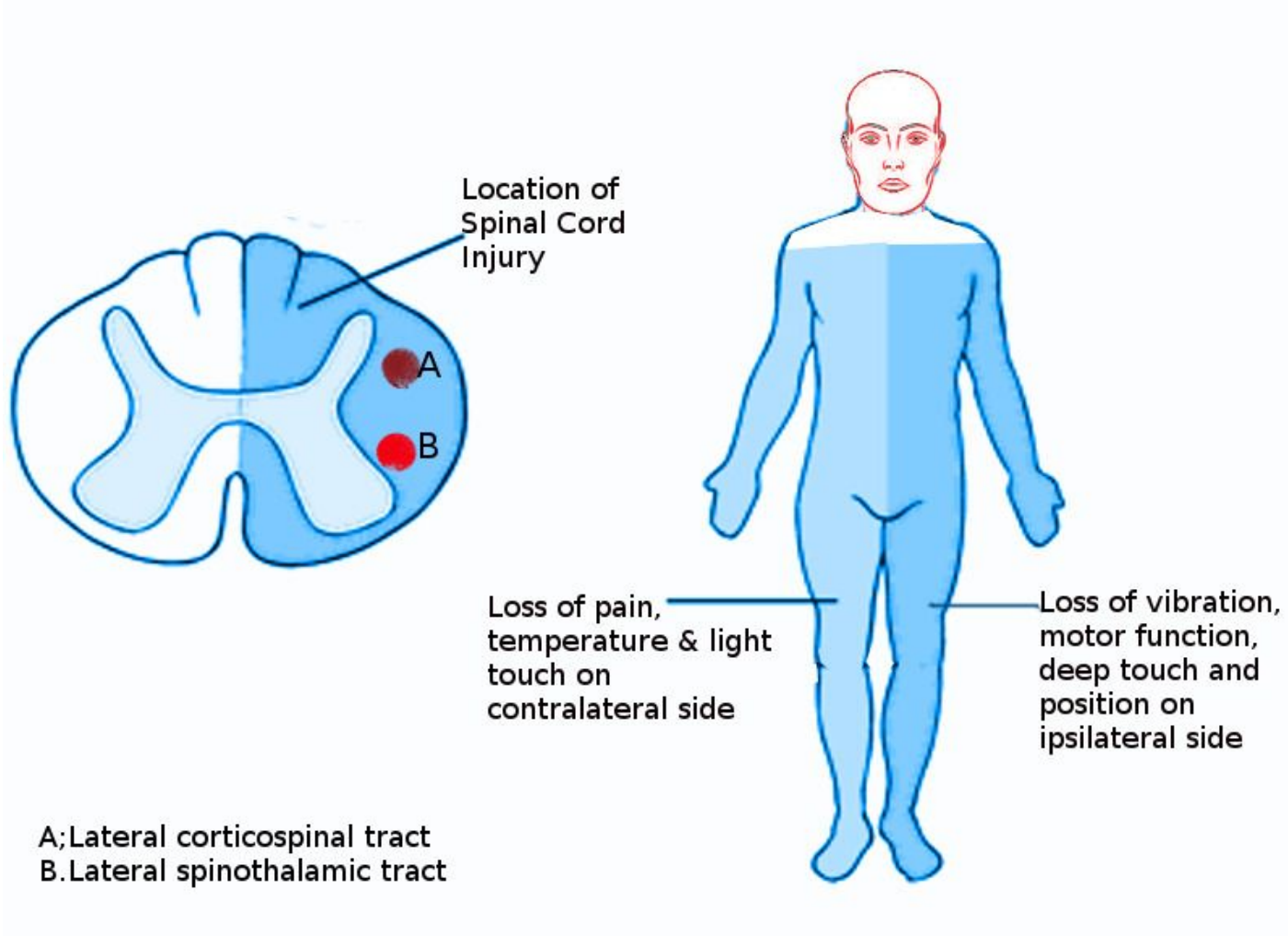




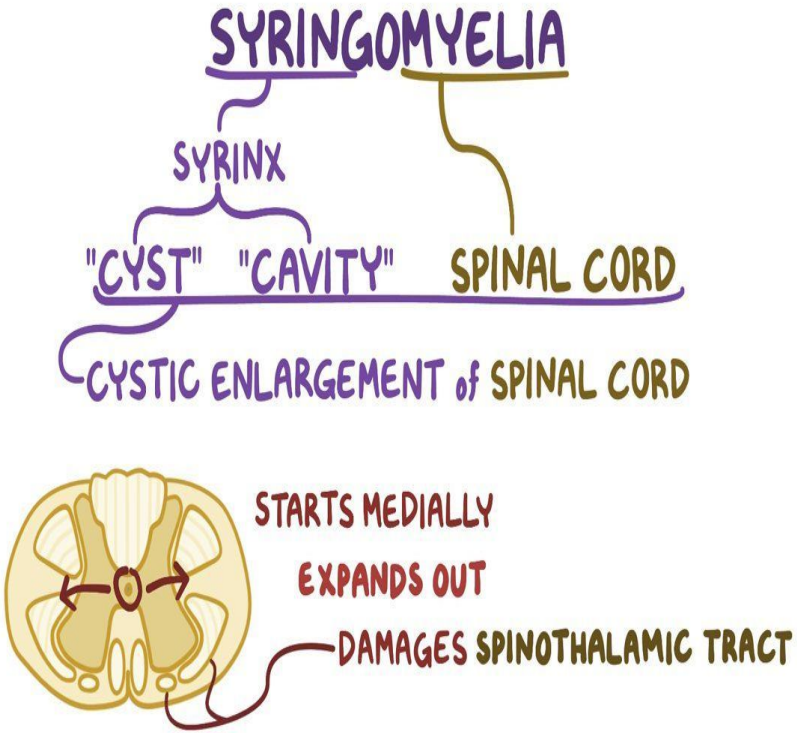
He was born at Port Louis, Mauritius, to an American father and a French mother. He attended the Royal College in Mauritius, and graduated in medicine at Paris in 1846. He then returned to Mauritius with the intention of practising there, but in 1852 he went to the United States.[3] There he was appointed to the faculty of the Medical College of Virginia where he conducted experiments in the basement of the Egyptian Building.

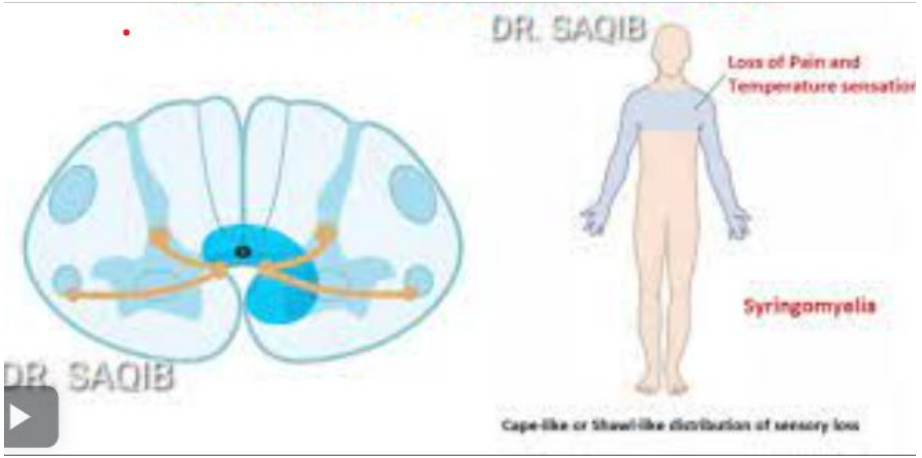
He was elected as a member of the American Philosophical Society in 1854.[4]




Location of Spinal Cord Injury
A
B
Loss of pain, temperature & light touch on contralateral side
Loss of vibration, motor function, deep touch and position on ipsilateral side
A;Lateral corticospinal tract
B.Lateral spinothalamic tract

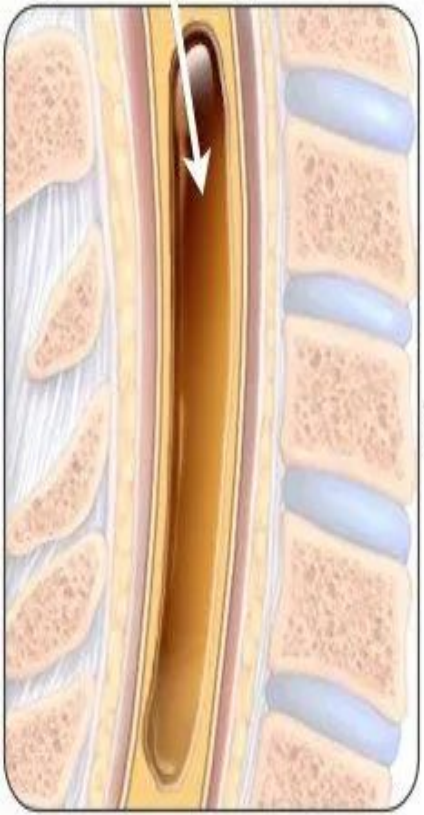
Syringomyelia





Syringomyelia

Cyst or **cavity** formation within the spinal cord
Expands and **elongates** over time

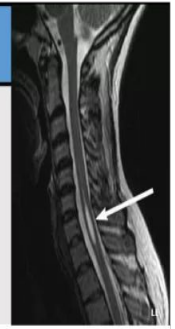


"Cape-like" distribution

loss of **pain** and **temperature sensation** in the **upper extremities**

preservation of **light touch** and **proprioception**

Syringomyelia



Causes	• Congenital development (or idiopathic)
	  • **Arnold-Chiari malformation**
	• Acquired
	  • Associated with tumors (intramedullary)
	  • Post-traumatic
	  • Arachnoiditis

Clinical Features	• Cavitation of the cord (usually cervical) • Bilateral loss of pain and temperature at the level of the lesion • As the disease progresses, there is muscle weakness; eventually flaccid paralysis and atrophy of the upper limb muscles due to destruction of ventral horn cells • **Horner's syndrome** due to involvement of cells in intermedio-lateral cell columns in first and second thoracic cord segments.
Diagnosis	• Myelogram may show widening of spinal cord (rarely done). • CT scan shows the widened cord. • **MRI is the most sensitive method.** It shows fluid-filled cavitation and dilated central canal.

Treatment	• **Surgical**
	  • Posttraumatic
	  • Decompression if indicated
	  • Surgically correct underlying condition
	  • e.g., posterior fossa decompression in Chiari I malformation

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Transverse Myelitis

- An acute, usually monophasic, demyelinating disorer affecting the spinal cord.
- It is usually thought to be post-infectious in origin
- Inflammation of the spinal across one level of the spinal cord.

Causes :

- Parainfectious
- Post-vaccinal (rabies)
- Systemic autoimmune disease
- Sarcoidosis
- Multiple sclerosis
- Neuromyelitis optica

Clinical feature :

- Weakness
- Sensory disturbance
- Bowel and Bladder dysfunction.
- Neuropathic pain
- Pain and temperature sensation diminished.

Investigation :

- MRI typically : cord swelling and gadolinium-enhancing lesions.
- CSF usually contains monocytes, protein increased, IgG index is elevated.

Management : • High-dose IV corticosteroids

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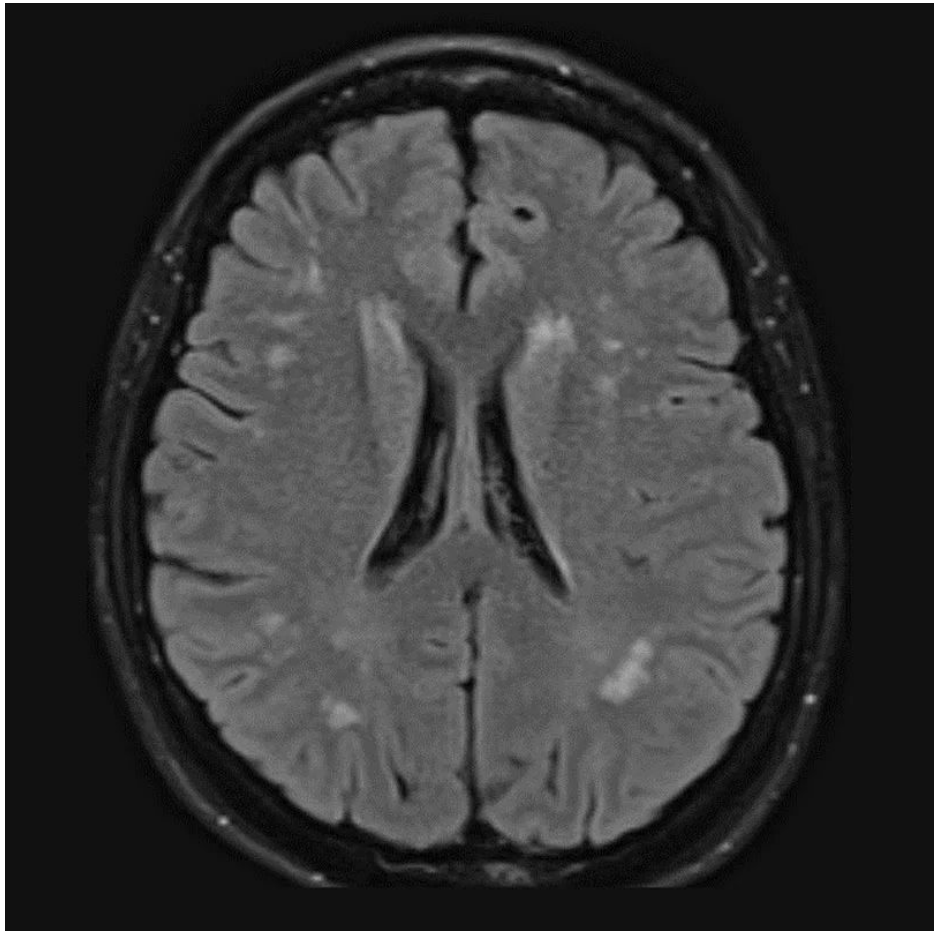
Diseases associated with TM

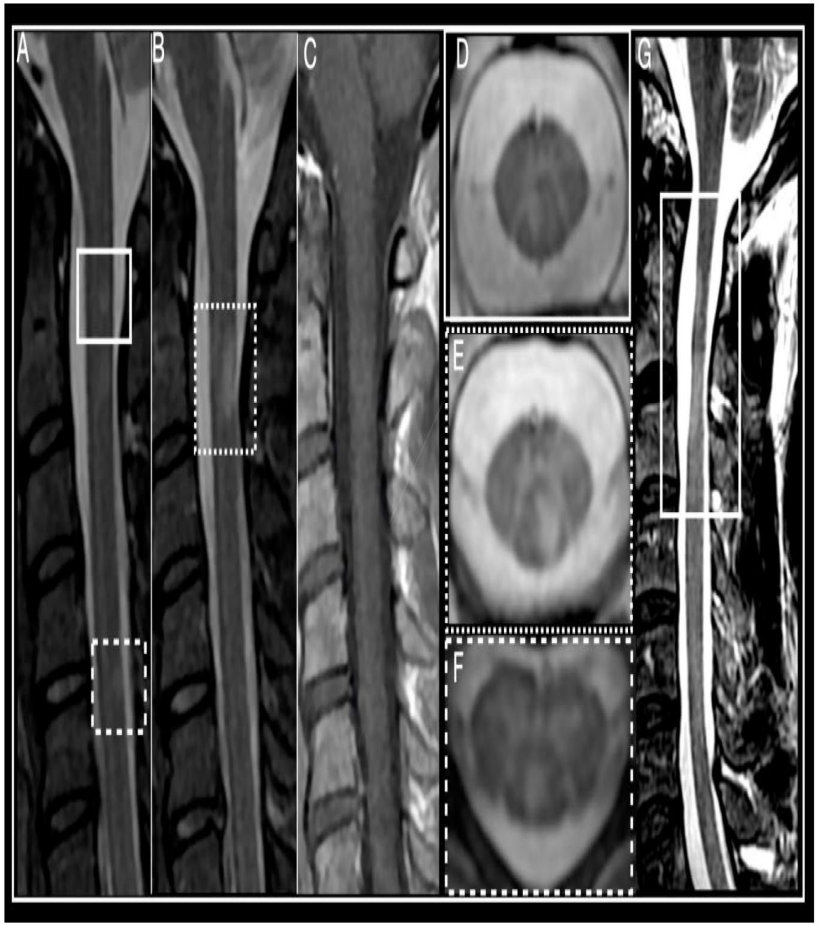
- *Parainfectious*
  - *Viral: HSV, herpes zoster, cytomegalovirus, Epstein-Barr virus, enteroviruses (poliomyelitis, Coxsackie virus, echovirus), human T-cell, leukemia virus, human immunodeficiency virus, influenza, rabies*
  - *Bacterial: Mycoplasma pneumoniae, Lyme borreliosis, syphilis, tuberculosis*
- *Postvaccinal - rabies, cowpox*
- *Systemic autoimmune disease*

  SLE, Sjogren's syndrome, Sarcoidosis
- *Multiple Sclerosis*
- *Paraneoplastic syndrome*
- *Vascular*

  Thrombosis of spinal arteries, Vasculitis secondary to heroin abuse, AV-malformation

MS





NMOSD

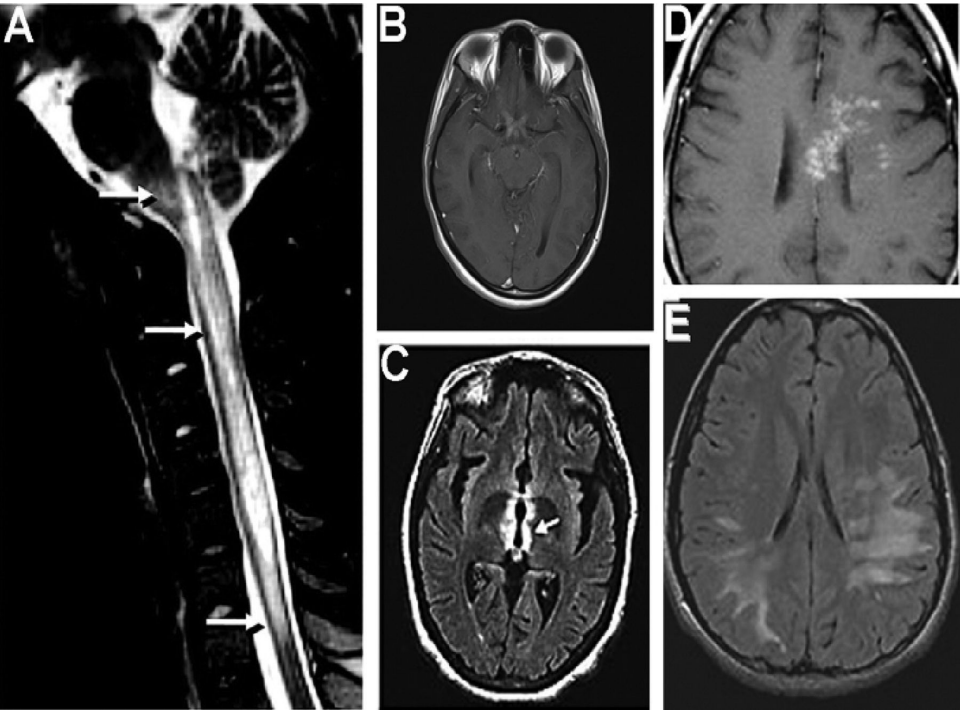


TABLE 10-2 **Features Differentiating Neuromyelitis Optica Spectrum Disorder From Multiple Sclerosis** *Continued from page 876*

Features	Neuromyelitis Optica Spectrum Disorder	Multiple Sclerosis
Laboratory findings		
Aquaporin-4 immunoglobulin G	Usually present	Absent
CSF cell count	Often very elevated, especially in the setting of relapse	>50 white blood cells very rare
CSF neutrophils and eosinophils	Often present	Usually absent
CSF protein	Often very elevated, especially in the setting of relapse	Usually normal, may be mildly elevated
CSF oligoclonal bands	Often absent (present in ≤25% of patients)	Usually present (in approximately 90% of patients)
CSF glial fibrillary acidic protein during relapse	Often very elevated	Normal or may be mildly elevated

CSF = cerebrospinal fluid; MRI = magnetic resonance imaging.

Management of patient with paraplegia

- MRI
- CT X-ray
- Blood investigation
  - aquaporin 4
  - antibody B12
  - vasculitic screen
  - PCR
- Spinal tap
- EMG Nerve Conduction Study
- IVMP
- IVMP + Steroids
- Plasma Exchange
- Immunosuppressants
- Biological antibodies
- Supportive Therapy
- Rehab

Update in Transverse Myelitis

- Glial cell therapy
  - OPC
- Astrosite antibodies
- Genetic studies to promote myelin protein
- Animal models
- New neuro imaging
- Brain devices

Paraparesis Mimics

Key Questions for Acute Paralysis:

- Has the patient had any recent illnesses, bites or stings, new foods, travel, or exposures?
- When was the onset of their symptoms?
- Does the patient have difficulty breathing, speaking, swallowing, double or blurry vision?
- What is the pattern of weakness? If it's progressive, is it ascending or descending? Proximal or distal?

Nerve conduction studies Nerve conduction studies demonstrate demyelination including slowed motor nerve conduction velocities, prolonged distal motor latencies, delayed F wave latencies (Table 2), and partial motor conduction block (at least 30 % to 50 % reduction in proximal amplitude) or abnormal temporal dispersion (prolongation of proximal motor response duration by

Guillain-Barré syndrome associated with SARS-CoV-2 infection: causality or coincidence?

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Clinical Case Reports Open Access WILEY

CASE REPORT

Guillain–Barre syndrome associated with hepatitis E virus infection: A case report

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Key Clinical Message

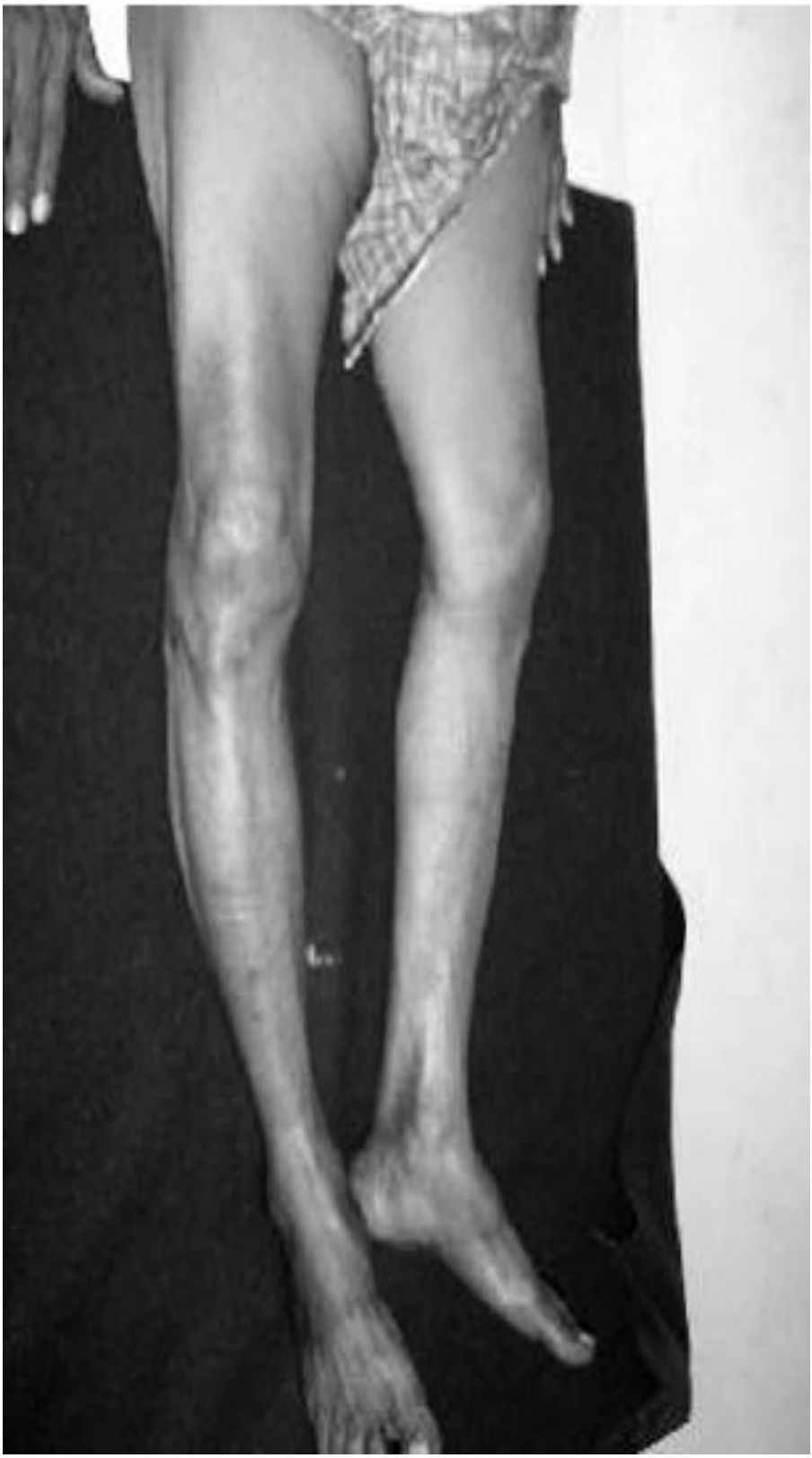
Hepatitis E virus (HEV) infection can be manifested with several neurological syndromes including GBS. Therefore, healthcare providers should consider HEV in their differential diagnosis for patients with neurological disorders.

Abstract

We report a case of Guillain-Barré syndrome associated with hepatitis E virus infection. The current case-report demonstrates diagnostic challenge to identify GBS case in a limited-resources country like Sudan. However, HEV infection should be highly suspected in patients with neurological manifestation with high liver enzymes.

KEYWORDS

critical care medicine, infectious disease, neurology, transdisciplinary one health strategy



Review > Mol Genet Metab. 2022 Dec;137(4):436-444. doi: 10.1016/j.ymgme.2021.06.006. Epub 2021 Jun 24.

Childhood-onset hereditary spastic paraplegia and its treatable mimics

Darius Ebrahimi-Fakhari [1], Afshin Saffari [2], Phillip L Pearl [3]

Affiliations + expand

PMID: 34183250 PMCID: PMC8843241 DOI: 10.1016/j.ymgme.2021.06.006

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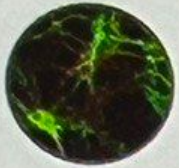
Abstract

Early-onset forms of hereditary spastic paraplegia and inborn errors of metabolism that present with spastic diplegia are among the most common "mimics" of cerebral palsy. Early detection of these heterogenous genetic disorders can inform genetic counseling, anticipatory guidance, and improve outcomes, particularly where specific treatments exist. The diagnosis relies on clinical pattern recognition, biochemical testing, neuroimaging, and increasingly next-generation sequencing-based molecular testing. In this short review, we summarize the clinical and molecular understanding of: 1) childhood-onset and complex forms of hereditary spastic paraplegia (SPG5, SPG7, SPG11, SPG15, SPG35, SPG47, SPG48, SPG50, SPG51, SPG52) and, 2) the most common inborn errors of metabolism that present with phenotypes that resemble hereditary spastic paraplegia.

The platform trial is a unique opportunity to move ALS biomark new outcome measures forward

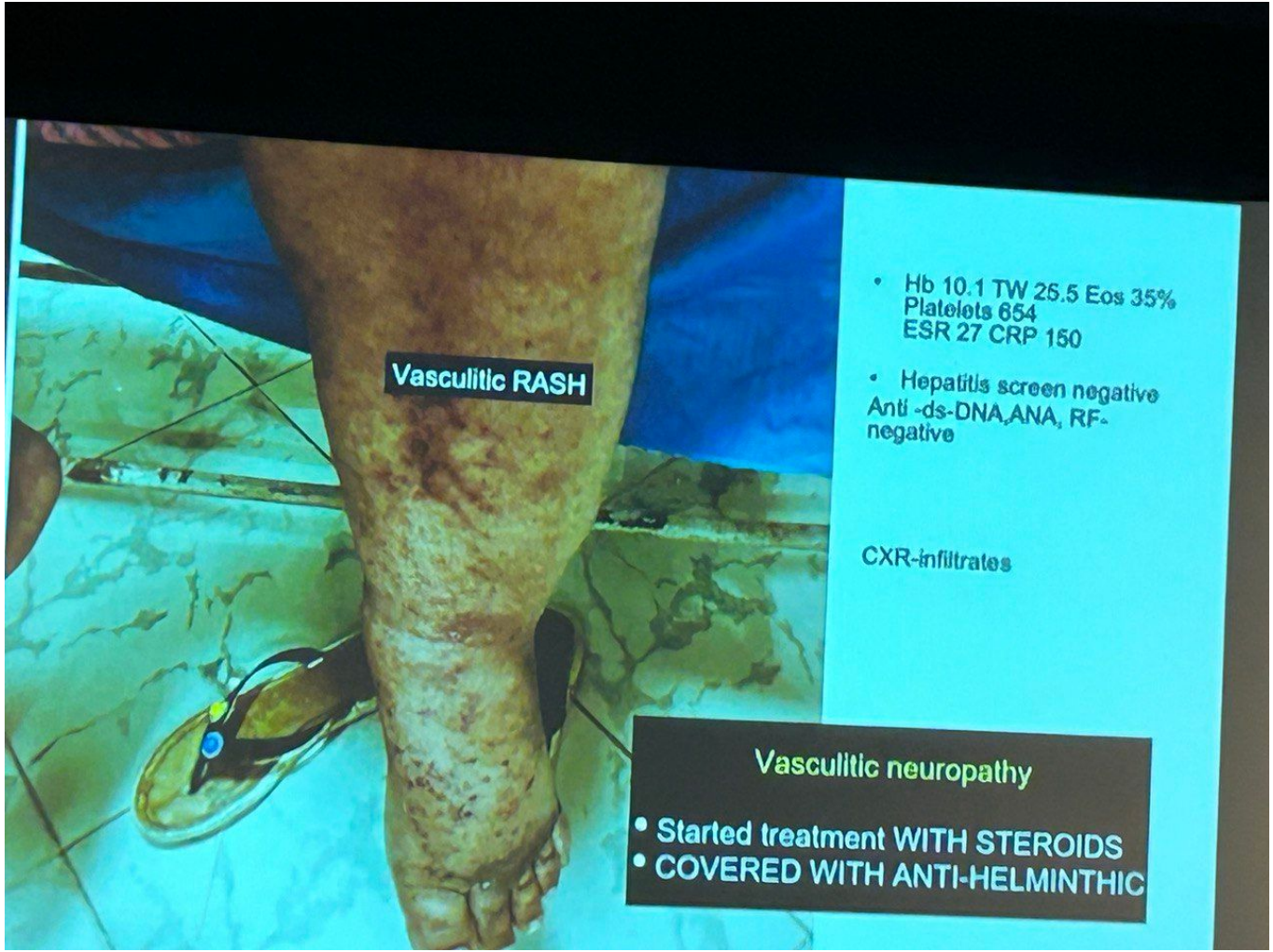
DNA – whole genome sequencing



Neurofilaments – for all regimens + regimen-specific biomarkers b

Home Spirometry – critical during the pandemic

Speech Analysis – emerging digital biomarker


Vasculitic RASH
• Hb 10.1 TW 25.5 Eos 35%
Platelets 654
ESR 27 CRP 150
• Hepatitis screen negative
Anti -ds-DNA,ANA, RF-
negative
CXR-infiltrates
Vasculitic neuropathy
• Started treatment WITH STEROIDS
• COVERED WITH ANTI-HELMINTHIC

Non-toxicologic causes of symmetric paralysis[3]

-GBS, including Miller Fisher variant

-Myasthenia gravis

-Hypokalemia

-Hyperkalemia

-Hypermagnesemia

-Encephalitis

-Hypokalemic periodic paralysis

-Lambert Eaton myasthenic syndrome

-Spinal cord compression or injury

-Transverse myelitis

-Poliomyelitis

-Polymyositis

Main Points:

- Patients with acute symmetric paralysis should undergo a detailed history and physical addressing exposures, travels, time of onset, patterns of weakness, presence of bulbar palsies, and reflexes.
- Peripheral nerve involvement often presents with decreased reflexes and abnormal sensory exams.
- Neuromuscular junction involvement often present with intact reflexes and sensation.
- Botulism is frequently misdiagnosed and early recognition is critical as patients often progress to respiratory failure that may be prevented with the early administration of botulinum antitoxin.[4]
- Tick paralysis is a very rare condition but similarly can progress rapidly to respiratory failure until the tick is identified and removed.[3]
- Other toxicologic sources of paralysis encompass a broad array of foodborne toxins, envenomations, and chemical exposures.[5]

Batracho-toxin[7]	Mixed effects. Venom extracted from poison dart frog skin of genus *Dendrobatidae* in South and Central America	Absent	Rapid total paralysis. Arrhythmias and cardiac arrest.	Clinical diagnosis.	Supportive treatment. Saxitoxin and tetrodotoxin prevent membrane depolarization but have significant side effects and are not an antidote.
Botulism[3,8]	Neuromuscular junction effects. *Clostridium botulinum* ingested in food or its spores are ingested by an infant, wound contamination, iatrogenic	Normal	Descending symmetric paralysis, normal sensation, early cranial nerve palsies, respiratory compromise.	CSF is normal. Formal diagnosis through toxin in serum, stool, or food or from *C. botulinum* growth in stool culture.	Supportive care. Intubation, airway support, Botulinum Antitoxin (H-BAT). Infants receive IV botulism immunoglobulin (Baby BIG)
Bungaro-toxin[9,10]	Mixed effects. Elapidae (Sea snakes, cobras, kraits, coral snakes)	Normal/ Decrease	Ascending symmetric paralysis. Minimal pain at the bite site with nausea, muscle rigidity, swallowing difficulty, blurry vision, myoglobinuria, respiratory compromise.	CSF is normal. Clinical diagnosis.	Immobilize limb. Urgent antivenom. Hyperkalemia, rhabdomyolysis, and renal dysfunction are common.
Conotoxin[9]	Mixed effects. Marine snails of genus *Conus*	Absent/ Decrease	Localized pain, numbness, ischemia at injection site. Paresthesias and weakness follow with cranial nerve palsies, possible respiratory and cardiac compromise.	CSF is normal. Clinical diagnosis. Envenomation site may not be visible.	Immobilize limb. Supportive care. Potential for cardiac dysrhythmias.
Curare[11]	Neuromuscular junction effects. Alkaloids extracted from the leaves of multiple plants in Central and South America	Absent	Motor weakness that rapidly becomes total flaccid paralysis and respiratory failure.	Clinical diagnosis.	Supportive treatment and early intubation. Anticholinesterase agents like pyridostigmine can reverse paralysis.
Konzo[12]	High dietary cyanogen consumption (mainly as linamarin) in cassava root.	Increase	Selective upper motor neuron damage, abrupt irreversible, non-progressive, and symmetrical spastic para/tetraparesis. Tropical ataxic neuropathy (TAN) is also associated with cassava consumption	Clinical diagnoses, can detect cyanogens in food samples.	Supportive treatment. Prevention with food preparation (soaking, sun drying, heap fermentation, grating plus roasting) and protein-rich diet. Draught or famine associated with poor preparation and cyanogen

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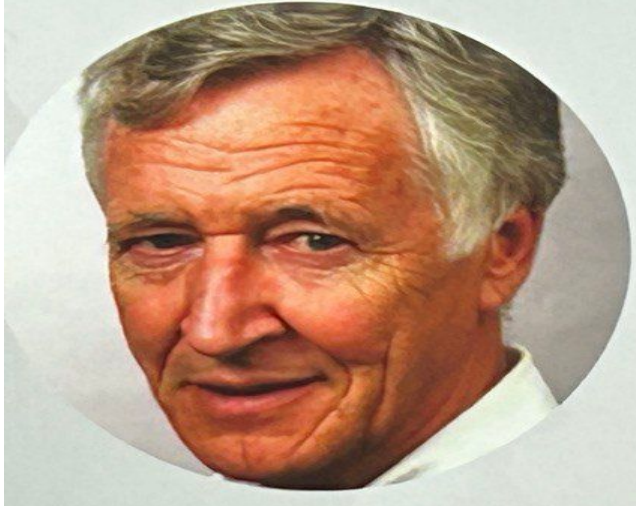

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WILLIAM HOWLETT

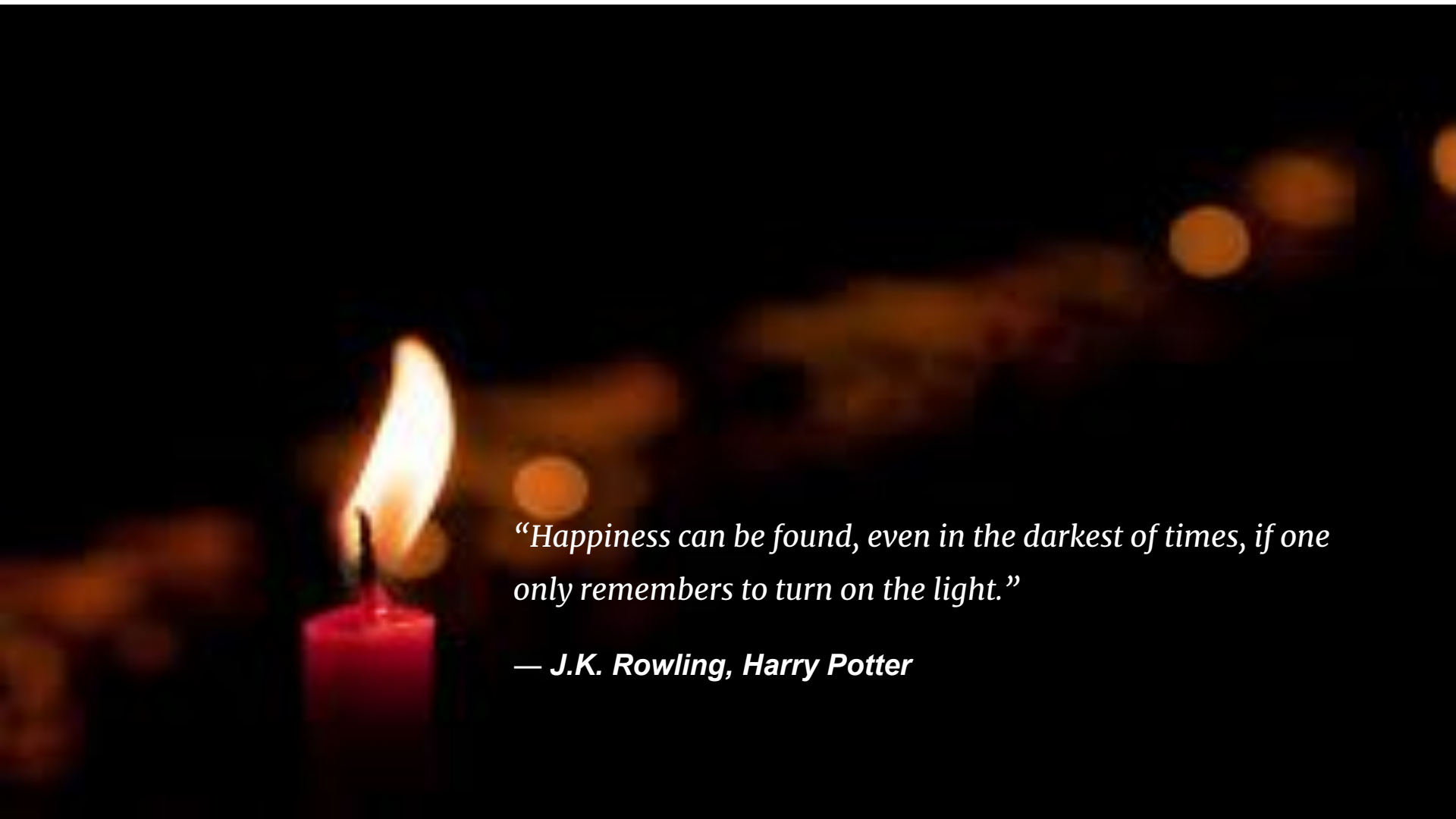
Tanzania

Within Tanzania, Nyerere has been termed the "Father of the Nation",[455] and was also known as *Mwalimu* (teacher).[456] He gained recognition for the successful merger between Tanganyika and Zanzibar,[457] and for leaving Tanzania as a united and stable state.[458] Molony noted that Nyrere was "often depicted as Tanganyika's *wunderkind*",[459] and is "remembered as one of Africa's most respected statesmen".[425]

Neurology MD Sudan Launched 2016


A team is
many hands & one mind.

He bends his back while the sun is blazing on him, but he is in love with the letter and pen. He writes his homework on his textbook. Yes, with knowledge, one rises to the heights of the peak


“Happiness can be found, even in the darkest of times, if one only remembers to turn on the light.”
— J.K. Rowling, Harry Potter