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A Case of Rapid Onset Tetraplegia in a Young Woman

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Abstract

A 31-year-old woman presented with generalised pain and weakness due to a severe axonal sensorimotor neuropathy rapidly worsening over two weeks. She described a history of transient joint symptoms, rash and hair loss six months prior to admission. A vasculitic process was considered, and blood tests taken post- intravenous immunoglobulin (IVIG) (administered for suspected Guillain Barré syndrome) noted strongly positive lupus and antiphospholipid antibody titres. She was diagnosed with severe multisystem lupus vasculitis involving the central and peripheral nervous system and initiated on cyclophosphamide and prednisolone, with notable improvement. We outline challenges faced in this patient's care, including making the diagnosis, interpreting autoantibody serology post-IVIG and identifying suitable immunosuppression regimens in a limited evidence base. It is important to be aware that peripheral neuropathy is an under-recognised presenting manifestation of neurological lupus.

Case presentation

A 31-year-old woman of Nigerian heritage presented to her local emergency department reporting severe generalised pain, and weakness and sensory loss in both lower limbs. She had no past medical or family history of neurological disease.

She reported ankle pain six months prior, subsequently developing swelling and a rash over her right ankle. These symptoms resolved over weeks but reoccurred in the left ankle a month later, again self-resolving.

Over six months prior to admission, she developed bilateral paraesthesiae in the hands and feet- gradually progressing to involve both arms and legs. Her rash also worsened, involving both legs and became ulcerated. In this period, she lost 10kg unintentionally, experienced worsening constipation and had an episode of marked alopecia.

Two weeks preceding admission, her symptoms deteriorated rapidly. She was able to walk into the emergency department. Pertinent initial examination findings were length-dependent weakness (MRC grade 2/5 power ankle plantarflexion and dorsiflexion; 4/5 knee flexion and extension; 5/5 hip extension), areflexia, impaired sensation to mid-shin and preserved upper limb power. She was initially treated for a suspected Guillain-Barré syndrome (GBS) with IVIG. A nerve conduction study demonstrated severe acute axonal sensorimotor neuropathy (Table 1). Cerebrospinal fluid (CSF) analysis was normal.

Despite IVIG initiation, she continued to rapidly decline and, within two weeks, became tetraplegic, only retaining minimal right shoulder movement. At this early stage, the degree of acute denervation on electromyography (EMG) was felt to be atypical for GBS. Factoring her antecedent 6-month history, a vasculitic neuropathy was considered and she was treated with intravenous methylprednisolone (IVMP).

She was transferred urgently to a neurosciences centre for specialist input. On arrival, neurological examination noted mild right shoulder weakness (4/5 power abduction and adduction) but marked tetraplegia (0/5 power throughout), global areflexia, absent plantar responses and globally impaired sensation across all modalities. Cranial nerves remained intact. She appeared unwell with cracked, ulcerated lips and multiple large, ulcerated rashes over both ankles with surrounding hyperpigmented granulation tissue (Figure 1).

Additionally, she had evidence of multi-system involvement with intermittent chest pain, sinus tachycardia and elevated troponin suggesting myopericarditis. Liver function tests showed persistent transaminitis with negative viral and autoimmune hepatitis serology. She also had a Coombs-positive autoimmune haemolytic anaemia.

Diagnosis

Blood for autoimmune serology was collected following IVIG administration and demonstrated positive autoimmune and antiphospholipid antibodies, with high ANA titres (Table 2).

MRI brain and spinal cord revealed subacute infarcts in the right cerebral peduncle and spinal cord (Figure 2). There was no evidence of malignancy or infection and CT thorax, abdomen and pelvis was normal.

A sural nerve biopsy showed widespread loss of myelinated axons with extensive fibrosis, neovascularisation and haemosiderin deposits. Additionally, there was perivascular perineurial inflammatory infiltrate with blood vessel wall infiltration. Overall, findings were consistent with an end-stage neuropathy with chronic/active vasculitic changes (Figure 3).

To summarise, this case features a subacute progressive neurological decline with clear multisystem involvement in a young woman. Neurophysiology revealed severe sensorimotor axonal neuropathy. Sural nerve biopsy showed perivascular infiltration and signs of longstanding post-vasculitic changes (scarring of the endoneurium with complete loss of myelinated fibres, neovascularisation and haemosiderin deposits). Autoimmune serology remained strongly positive despite potential IVIG-related variation. Multiple bilateral presumed subacute infarcts were evident on imaging, with otherwise normal CSF and systemic imaging.

Ultimately, the unifying diagnosis made was severe lupus-associated vasculitis and antiphospholipid syndrome (APLS) with extensive central and peripheral nervous system involvement and multi-system manifestations.

Management

Initiating appropriate immunosuppression was central to management. She completed three days of IVMP followed by high-dose oral prednisolone. Intravenous cyclophosphamide was then initiated following the CYCLOPS regimen (Table 3).

Given her strongly positive antiphospholipid antibodies and radiological evidence of subacute infarcts, therapeutic anticoagulation was commenced following multidisciplinary discussion. Decision complexity centred around infarction aetiology (whether they were related to vasculitic processes or secondary to APLS and requiring anticoagulation with warfarin).

Clinical course

Prompt immunosuppression initiation with cyclophosphamide and prednisolone resulted in marked neurological and systemic improvements. After five cycles of cyclophosphamide, her cardiac symptoms resolved, liver function tests normalised, and neuropathic pain improved.

She regained significant proximal limb strength and truncal control and was transferred to a neuro-rehabilitation facility.

Discussion and complexities

SLE is a multisystem disorder and lupus vasculitis- a rare but severe manifestation- involves inflammatory damage and vessel wall necrosis of small-to-medium vessels (1). It is associated with significant morbidity and mortality with multi-organ involvement, including skin, kidneys, and central and peripheral nervous system. Table 3 outlines more information about other multisystem manifestations.

Lupus vasculitis management is not well-defined due to its rarity and clinical heterogeneity. Current treatment strategies- largely extrapolated from lupus nephritis treatment- involve aggressive immunosuppression to reduce immune-complex deposition and vascular inflammation.

APLS, diagnosed in 10-15% of SLE patients, is characterised by antiphospholipid antibodies such as lupus anticoagulant, anticardiolipin, and anti-β2 glycoprotein-I. It is associated with a high risk of venous and arterial thromboses. APLS-associated strokes are primarily thrombotic due to hypercoagulable states, rather than vasculitic infarcts (2).

Clinical complexities arose in every element, from diagnosis to management. Outlined below are the most pertinent challenges we faced;

1. Making the initial diagnosis

GBS is the commonest cause of a rapidly progressive severe generalised neuropathy and was the initial working diagnosis. It is important to remember that there are other causes of acute-onset peripheral neuropathy that can mimic GBS including vasculitis, pan-neurofascin antibody mediated neuropathy, toxins and metabolic diseases such as dry beri-beri. The clinical clues that prompted further investigation into alternative causes was the longer history of neuropathic pain, skin ulcers, weight loss and the profuse fibrillation potentials on EMG performed on admission suggesting a more chronic process than GBS, including the acute motor and sensory axonal variant (AMSAN).

Although lupus vasculitis was suspected based on the clinical presentation and positive serology, diagnostic criteria were not met. The most recognised SLE diagnostic guidelines come from the 2019 European League Against Rheumatism/American College of Rheumatology (EULAR/ACR) classification criteria where formal SLE diagnoses require a

significantly positive ANA result, evidence of at least 1 systemic clinical feature and the presence of immunological markers equalling at least 10 points (3).

Whilst the criteria recognises some neuropsychiatric symptoms (namely seizures, psychosis and delirium), it does not explicitly include neuropathy as a neurological manifestation despite studies demonstrating relatively high prevalences in SLE from 7%-14% (4,5). Similarly, lupus vasculitis, although a recognised SLE manifestation, is not part of the diagnostic criteria. Clinicians should be aware that neuropathy and vasculitis are uncommon but reported manifestations of SLE, and caution against rigid application of EULAR/ACR diagnostic criteria. Additionally, it is important to investigate broadly and exclude alternative aetiologies.

Commonly identified manifestations of peripheral neuropathy in SLE include length-dependent polyneuropathy, mononeuritis multiplex and cranial neuropathies. Early recognition of peripheral nervous system involvement in SLE is important, given its associations with higher SLE disease activity and greater impact on quality of life (4,5).

2. Interpretation of autoantibody titres post-IVIG infusion

It is widely understood that serum antibody titres can be misinterpreted after IVIG administration. IVIG is derived from pooled plasma of thousands of donors, and autoantibody testing after IVIG infusions can therefore result in false positive results.

A core diagnostic challenge faced was that primary autoimmune screening tests were taken after IVIG infusion. Consequently, despite positive autoantibody results, interpretation was challenging.

A study by Bright *et al.* assessing autoantibody presence from different IVIG products found most autoantibody tests were negative at physiological IVIG concentrations. Any false positive results for other autoantibodies were only detected at non-physiological concentrations (6).

IVIG half-life and the timing of serum testing post-IVIG is an important consideration. Miyamoto *et al.* reviewed autoantibody concentrations from different IVIG preparations several weeks after infusion and demonstrated that anti-cardiolipin and anti-dsDNA antibodies concentrations declined, steeply by day five post-IVIG and gradually thereafter (7).

These findings underscore key elements of autoimmune serology interpretation post-IVIG- the importance of correlating clinical symptoms to antibody results, the antibody titre itself and, if still unsure, the benefit of repeating serological testing at regular intervals after IVIG.

This patient demonstrated strongly positive titres initially after IVIG (Table 1) with a clinically consistent history. Repeat autoimmune serologies two weeks and one month after IVIG infusion revealed persistently positive thrombophilia screens, anti-dsDNA level and positive ANA. In hindsight, the ANA and dsDNA antibody titres were too high to be explained by the recent IVIG infusion.

3. Identifying a suitable immunosuppression regime

2024 EULAR recommendations support cyclophosphamide use in SLE if life-threatening/features of end-organ damage are present (3). There is a lack of specific

guidelines for cyclophosphamide use in lupus vasculitis given its rarity. Management is often guided by clinical experience.

Most current cyclophosphamide guidelines come from studies evaluating lupus nephritis treatment (8), and total cyclophosphamide doses can vary (Table 4).

Regimen choices should be carefully made, considering disease severity, holistic clinical status and baseline biochemistry. Given her rapid neurological decline and multisystem involvement, we opted for the CYCLOPS regime due to its higher total dose and the severity of her disease.

4. Cyclophosphamide and fertility considerations

Given its well-established gonadotoxic nature, cyclophosphamide initiation in women of childbearing age needs family-planning consideration. Cyclophosphamide is associated with amenorrhoea, permanent ovarian failure and teratogenicity (9). Although fertility optimisation options exist through gonadotrophin-releasing-hormone agonists (GNRHa) and oocyte cryopreservation, they need balancing against clinical stability.

Our main challenge was that this patient was too unwell to await cryopreservation but wanted children. Gynaecology and fertility team reviews concluded that her cumulative cyclophosphamide dose posed low gonadotoxicity risks, so GNRHa risks would outweigh benefits.

Clinicians need to recognise the adverse fertility effects of cyclophosphamide and facilitate early patient discussions. Gynaecology teams should be consulted to consider fertility preservation where appropriate (10).

This case highlights significant complexities of acute lupus vasculitis with neurological-predominant features. The co-occurrence of SLE, vasculitic neuropathy, APLS and spinal and cerebral infarctions represent complex clinical challenges from the lack of robust evidence-base. Clinicians should be aware of SLE-associated neurological symptoms and the importance of holistic, multidisciplinary approaches in navigating uncertainty in investigations and management.

Key points

1. Neuropathy and vasculitis are rare but severe manifestations of SLE and are associated with significant morbidity- neuropathy is, however, not currently included in the EULAR/ACR diagnostic criteria as a neurological manifestation of SLE
2. In patients with SLE and peripheral neuropathy, this case illustrates the importance of looking for evidence of CNS involvement, investigating for the coexistence of APLS and SLE and considering early anticoagulation to prevent brain or spinal infarction
3. Strongly positive autoimmune serology post-IVIG administration is unlikely to be attributable to IVIG

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Appendix

Table 1: Admission nerve conduction study and EMG. Lat- latency; Amp- amplitude; CV- conduction velocity

Sensory						
	Left			Right		
	Lat - ms	Amp - µV (tilt)	CV – m/s	Lat - ms	Amp - µV (tilt)	CV – m/s
Median (F3- wrist)				2.17	22.8	52.5
Ulnar (F5- wrist)				1.75	10.1	53.6
Radial (wrist- snuff box)				1.46	24.3	55.4
Superficial peroneal (shin- ankle)					Absent	
Sural (calf- ankle)		Absent			Absent	
Soleus H wave		Absent			Absent	

Motor								
	Left				Right			
	Lat - ms	Amp - mV (p-p)	CV – m/s	F-M Lat- ms	Lat - ms	Amp - mV (p-p)	CV – m/s	F-M Lat- ms
Motor median								
Wrist-APB					3.87	0.2		Absent
Elbow- wrist						0.23	48.4	
Motor ulnar								
Wrist-ADM					3.10	0.22		Absent
Elbow- wrist						0.11	44.8	
Lateral popliteal- tibialis anterior								
Tibialis anterior	3.25	0.34		16.6	3.27	0.22		17.1
Motor lateral popliteal								
Ankle- EDB		No response				No response		
Motor medial popliteal								
Ankle- AH		No response				No response		
EMG								

Right and left tibialis anterior and gastrocnemius	Profuse fibrillation potentials and positive sharp waves a rest, no voluntary unit recruited
Right vastus medialis	No spontaneous activity, on volition an excess of polyphasic motor unit potentials mostly noted interference pattern mild to moderately reduced.

Table 2: Pertinent autoimmune screen, with reference ranges, taken post-IVIG and follow up serology off IVIG- ten days, and one month after completion

Test name (reference range, if present)	Post-IVIG results	Ten days after IVIG	One month after IVIG
ANA HEp 2 cells	Strong positive 1/1280 - 1/2560, homogenous pattern	Strong positive 1/5120, homogenous pattern	Strong positive 1/1280, homogenous pattern
Anti-dsDNA antibodies (0-9.9 IU/mL)	127 IU/mL	135 IU/mL	42 IU/mL
ANCA	Positive p-ANCA	Atypical p-ANCA	-
Myeloperoxidase antibodies (0-5.97 IU/mL)	2.0 IU/mL	0.3 IU/mL	-
Anti-cardiolipin IgG (0-9.9 GPLU/mL)	304 GPLU/mL	227.6 GPLU/mL	-
B2GP1 antibodies (0-6.9 U/mL)	139 U/mL	404.6 U/mL	287.6 U/mL
Lupus anticoagulant/DRVVT (0-1.2)	Positive; 1.58	Positive; 2.40	Positive; 1.64
C4 (0.1-0.4 g/L)	0.13 g/L	0.14 g/L	0.2 g/L
Rheumatoid factor	Positive	-	-
Lactate dehydrogenase (0-250 u/L)	316 u/L	272 u/L	273 u/L

Table 3: General overview of some other multisystem features in SLE pertinent to this patient

System involved	Relative frequency	Clinical details
Cardiovascular	Common	- Can affect all components of the heart including the vasculature and conduction system - Pericarditis is the most frequent cardiac manifestation - Accelerated atherosclerosis and premature coronary artery disease
Hepatic	Rare	- Liver dysfunction can range from mild to severe - Mild forms include transient elevations in liver enzymes - Severe manifestations include lupus hepatitis, which may present with jaundice and hepatomegaly - Most common hepatic finding is non-alcoholic fatty liver disease, often related to corticosteroid therapy
Haematological	Common	- Manifestations include anaemia, thrombocytopaenia or leucopaenia - Most common cause of anaemia is anaemia of chronic disease, but haemolytic anaemia is a common complication - The presence of haemolytic anaemia can indicate a more severe form of SLE

Table 4: Outline of commonly used IV cyclophosphamide regimens, with reference ranges

Name	Regimen details	Maximum possible cumulative cyclophosphamide dose
NIH Regimen	500-1000mg/m^2 IV 4 weekly administered monthly for 6 months	6g over 24 weeks
Euro-Lupus (low dose) Regimen	500mg IV administered 2 weekly for 6 doses	3g over 12 weeks
CYCLOPS Regimen	15mg/kg (maximum 1500mg) 2/3 weekly for 10 doses	15g over 27 weeks

Figure 1: Images of ankle rashes on admission. Left ankle (A) and right ankle (B).

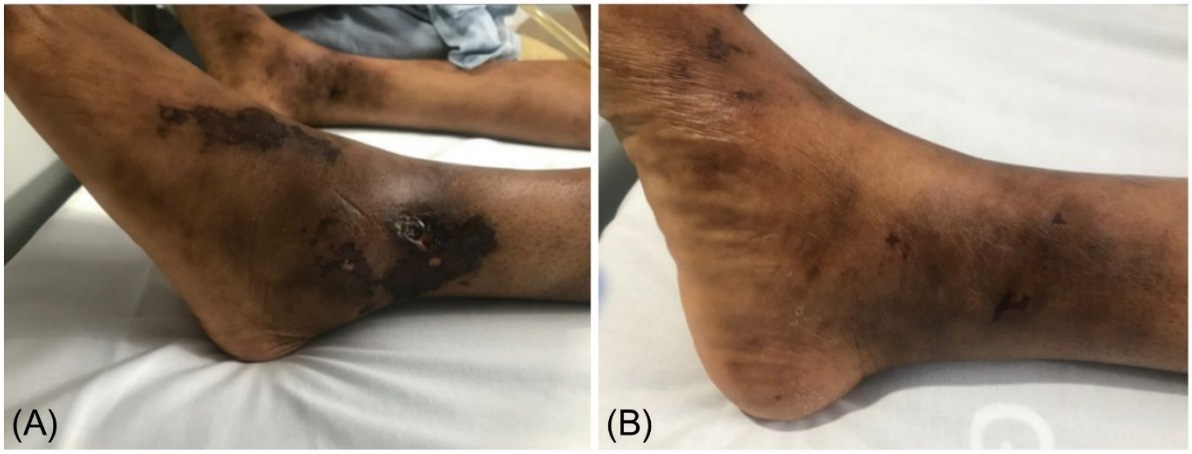


Figure 2: MRI head imaging demonstrating right cerebral peduncle and medullary lesions showing high DWI signal without enhancement or correlating ADC signal, suggestive of subacute chronicity. MRI whole spine demonstrating a T4/5 intramedullary lesion with restricted diffusion without enhancement. (i) MRI head T2 weighted; (ii) MRI head DWI; (iii) MRI head ADC; (iv) MRI spine T2 weighted (v) MRI spine T2 weighted.

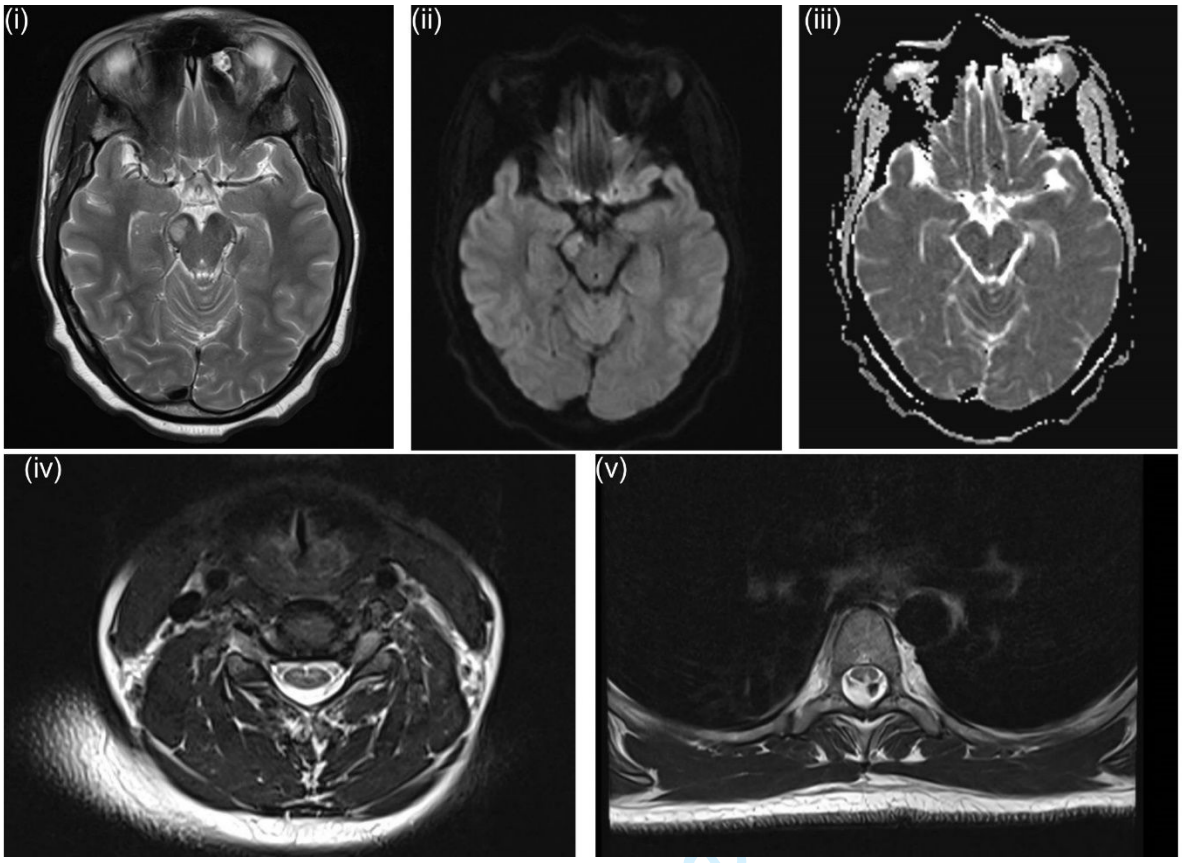
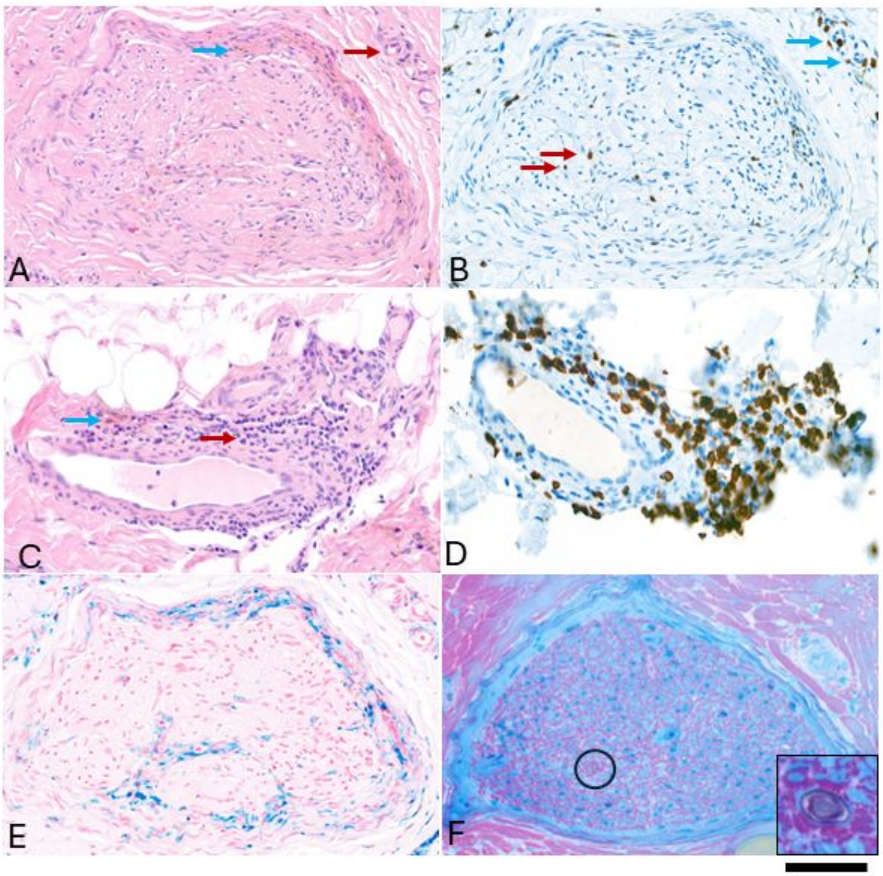
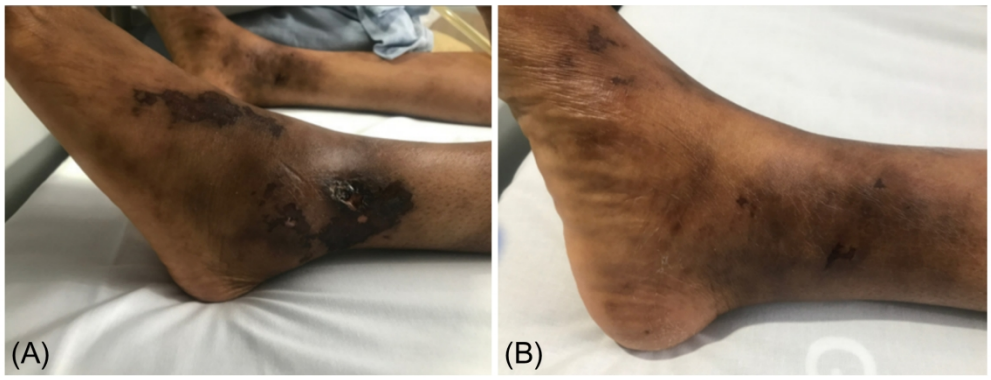


Figure 3: Sural nerve biopsy. (A) Haematoxylin and Eosin (H&E) of a transverse section of a collagenised nerve fascicle showing an end-stage neuropathy with fibrosis of the perineurium, with haemosiderin pigment (blue arrow) and neovascularisation in the epineurium (red arrow). (B) CD3 immunostaining shows endoneurial (red arrows) and epineurial T-lymphocytes (blue arrows). (C) H&E staining of an epineurial blood vessel with focal haemosiderin pigment (blue arrow), perivascular T-lymphocytes infiltrating the blood vessel wall (red arrow), and (D), positive for CD3 by immunostaining. (E) Perls iron stains highlights haemosiderin deposition (blue pigment) in the three nerve biopsy compartments, endoneurium, perineurium and epineurium. (F) Resin sections show the loss of all myelinated fibres, as well as a rare degenerating axon (circle and inset). Scale bar 100μm (inset 10μm)





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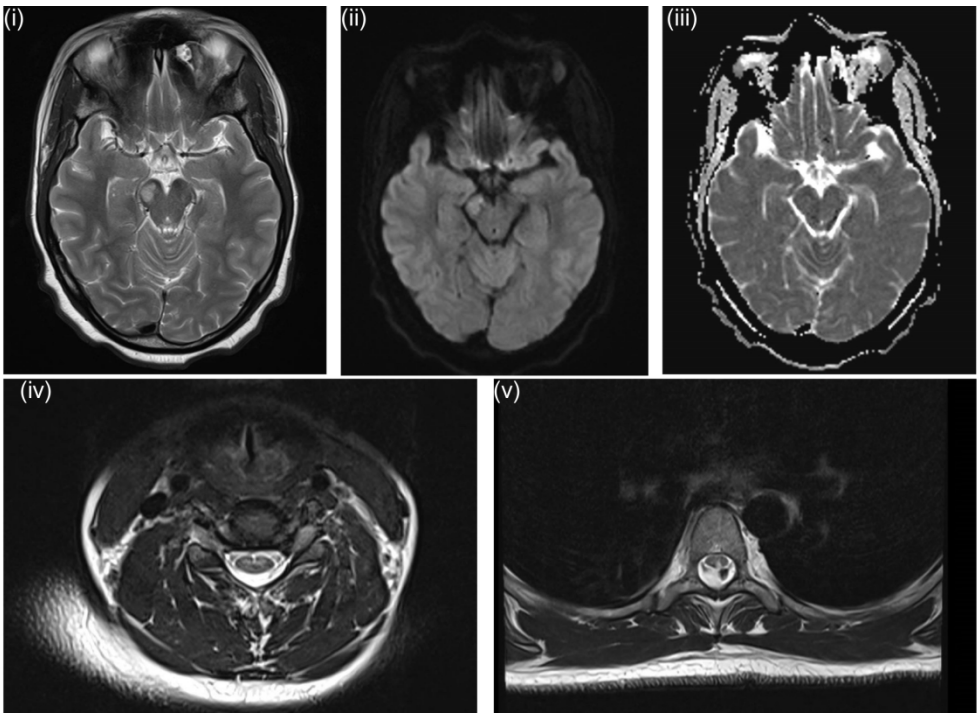

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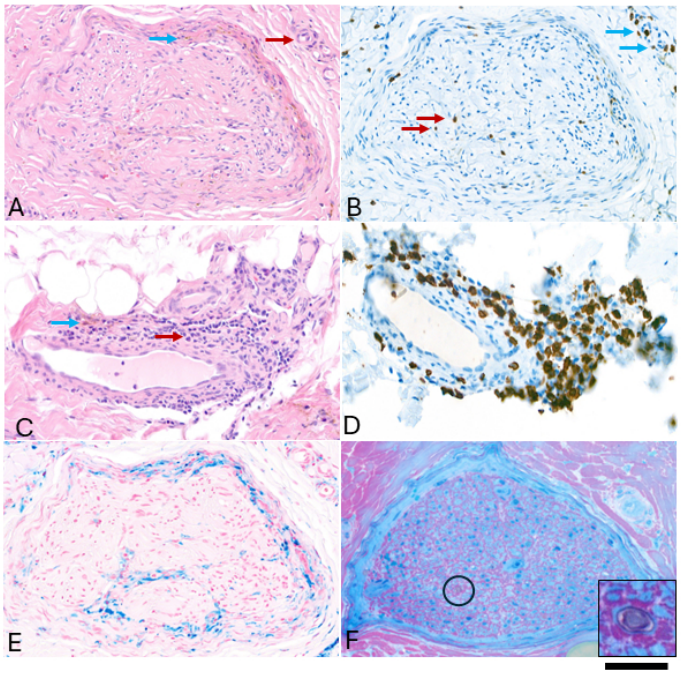

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