

ALL THAT BURNS IS NOT SMALL FIBER NEUROPATHY!

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Background and Aim:



Even in this era of Artificial Intelligence, **accuracy in diagnosis** depends on **meticulous history taking and detailed clinical examination**. In high-pressure clinical environments, subtle yet critical findings may often be missed, contributing to growing costs and at times, catastrophic consequences. We aim to describe how a detailed clinical review established an accurate diagnosis in a patient with a **clinical phenotype of 'small fibre neuropathy'**, when extensive laboratory investigations, electrophysiological studies and neuroimaging were inconclusive.

Method:



Case review of a **25-year-old lady** who presented with painful paresthesias of the lower limbs.



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Case Report:



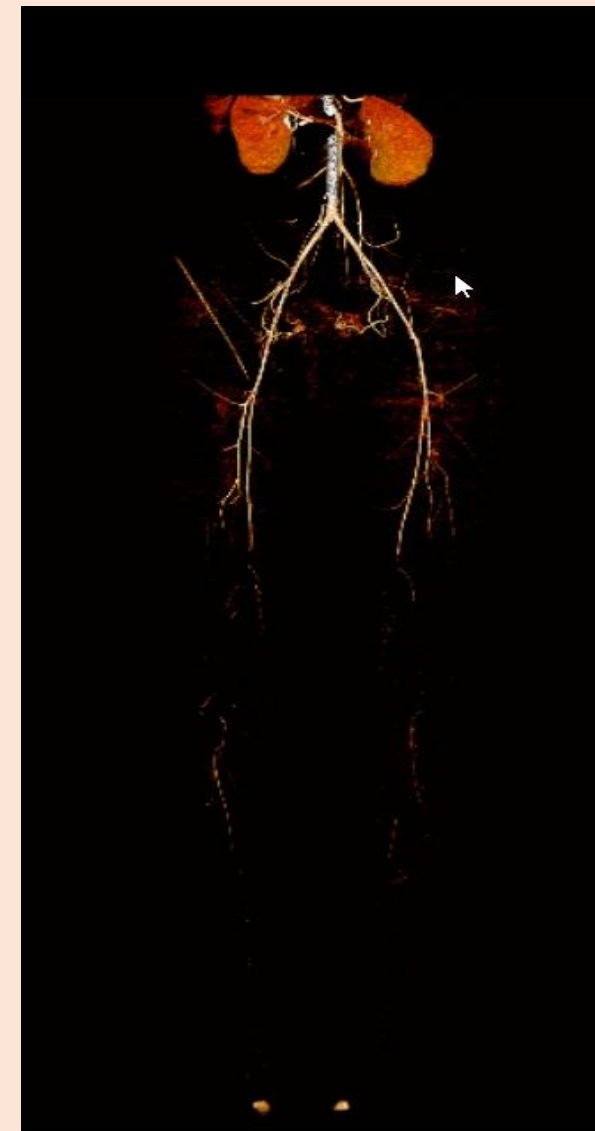
The patient is a **25 year old lady** who presented with **complaints of recurrent episodes** of intermittent pain, numbness, tingling, and burning paresthesias of both lower limbs which ascended from toes to mid-leg over the past 6 months. Neurological examination revealed impaired touch and pin sensations below mid-leg and an antalgic gait.

Electroneuromyography, MRI spine, neurography and brain screening were normal and she was diagnosed with small fibre neuropathy. Workup for the underlying aetiology, such as hemogram, hepatic, renal and thyroid function tests, blood sugar, lipid profile and vasculitic profile was negative. Initially, there was a partial response to gabapentin, but later the symptoms became continuous and disabling, such that she was unable to stand/walk due to the pain.

Presentation at NIMHANS:



In view of lack of improvement of symptoms, she presented to NIMHANS for further management. A clinical review and detailed general physical examination **revealed cool peripheries and absent distal pulses in both lower limbs with no gangrenous skin changes.** Neurological examination was unchanged. Hence, arterial Doppler and CT angiography was done which **confirmed the presence of complete occlusion and non-visualisation of lower limb vessels distal to the femoral arteries.**



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Diagnosis:



But why should a patient have limb ischemia at this age? **The answer was provided by the history** – she had been **consuming daily ergotamine-caffeine combination (over the counter medication) for her chronic daily headache**. She had also recently started to consume Sumatriptan for the last 2-3 months and Azithromycin every 15 days for the past 6 months for ‘sinusitis’. Hence the final diagnosis was **CHRONIC ERGOTISM INDUCED VASCULAR CLAUDICATION** and she was referred to Vascular surgery team for angioplasty and stenting.

Discussion:



Chronic ergotism or **St. Anthony's fire**, though a rarity these days, can occur with **unsupervised use of ergot alkaloids**, particularly in combination with **CYP3A4 inhibitors**, i.e., sumatriptan and azithromycin (inhibition of CYP3A4 increases ergotamine levels). **Classical skin changes of vascular insufficiency may be absent** and masquerade as small fibre neuropathy.

Conclusion:



Thus, despite great technological advances, **diagnostic accuracy** depends not only on data analysis but also on the **clinician's ability to extract, prioritize, and interpret the right data**- a skill machines have yet to master and which still remains the domain of the clinician. **Meticulous history taking (including drug history) and examination remain paramount.**



References:

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