# Primary Cerebral Nocardiosis Without Focal Neurological Deficit in a Patient of Focal Segmental Glomerulosclerosis: A Case Report

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## ABSTRACT

*Nocardia farcinica* is an aerobic opportunistic gram-positive bacterium. It is known to infect mainly immunocompromised hosts, primarily affecting lungs and skin, leading to invasive lung disease and disseminated infection. It has a high relapse and mortality rate. However, it can also affect immunocompetent individuals. This case report underscores the importance of keeping a high index of suspicion for cerebral nocardiosis, without any pulmonary or skin involvement, in an immunocompromised patient with raised intracranial pressure and headache. It also highlights the importance of multidisciplinary collaboration for infectious disease management.

**Key Words:** *Nocardia*, *Cerebral abscess*, *Focal segmental glomerulosclerosis*, *Immunosuppression*.

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## INTRODUCTION

*Nocardia farcinica* is an aerobic, filamentous opportunistic actinomycete primarily found in soil, salt, and freshwater, from where it can be transmitted *via* aerosol inoculation, or through a disrupted skin barrier.[1] It primarily infects the lungs in approximately two-thirds of cases.[2] Its hyphal structure favours invasive nocardiosis from the point of entry to other organs such as the brain, kidneys, eyes, joints, and bones, *via* a haematogenous route.[3] About 30% cases of invasive *N. farcinica* are documented to involve the central nervous system, which makes up to 2% of all cerebral abscesses.[4] The mortality in cerebral nocardiosis ranges from 33% for single lesions to 66% for multiple lesions.[4] Here, we present a case of a 46 year male who developed primary cerebral nocardiosis on a background of focal segmental glomerulosclerosis (FSGS). This case report highlights the diagnostic and therapeutic difficulties, due to a deranged renal profile, in a resource-limited country. This case report was prepared in accordance with the surgical case report (SCARE) guidelines.[5]

## CASE REPORT

A 46-year male, known hypertensive for one year, presented to the Nephrology Outpatient Department (OPD) with complaints of generalised body swelling, which was gradual in onset and progressive, associated with frothy urine for six months.

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There was no history of fever, productive cough, shortness of breath, chest pain, diarrhoea, vomiting, haemoptysis, haematuria, burning micturition, joint pains, oral or digital ulcers, jaundice, alopecia, rash, and nephrotoxic medicine intake. He took amlodipine 5 mg for hypertension.

His initial investigations showed haemogloblin (Hb) of 12.8 g/dl, white blood cells (WBC) count of 8200/µl, platelets of 230000/µl, serum creatinine of 3.1 mg/dl, urea of 92 mg/dl, and albumin of 2.6 g/dl. A complete urine examination showed 3+ proteinuria and a 24-hour urine analysis quantified it as 3.1 g. Other investigations, which included liver function tests, C3, C4 levels, antinuclear antibody (ANA), HbsAg, anti-HCV, anti-HIV 1/2, HbA1c, ultrasound abdomen, ECG, and echocardiography, were normal. A renal biopsy was done to diagnose the underlying cause of proteinuria. It showed FSGS, not otherwise specified (NOS) type, with moderate interstitial fibrosis, moderate tubular atrophy, and moderate interstitial nephritis. Consequently, he was given a trial of steroids. Tablet deltacortil 60 mg was given in 2 divided doses daily for four months, but he did not show improvement in the degree of proteinuria. He was labelled as having the steroid-resistant disease. Therefore, he was started on tacrolimus 0.5 mg twice daily, along with tapering steroids.

Unfortunately, after 1.5 months of treatment with tacrolimus, the patient started complaining about new-onset, dull left-sided frontotemporal headache associated with photophobia, phonophobia, and vomiting, but the headache had no diurnal variation or postural effect. It was not relieved with any medication. There was no accompanying history of fever, seizures, focal neurological deficit, or altered sensorium. Figure 1 shows a timeline of events.

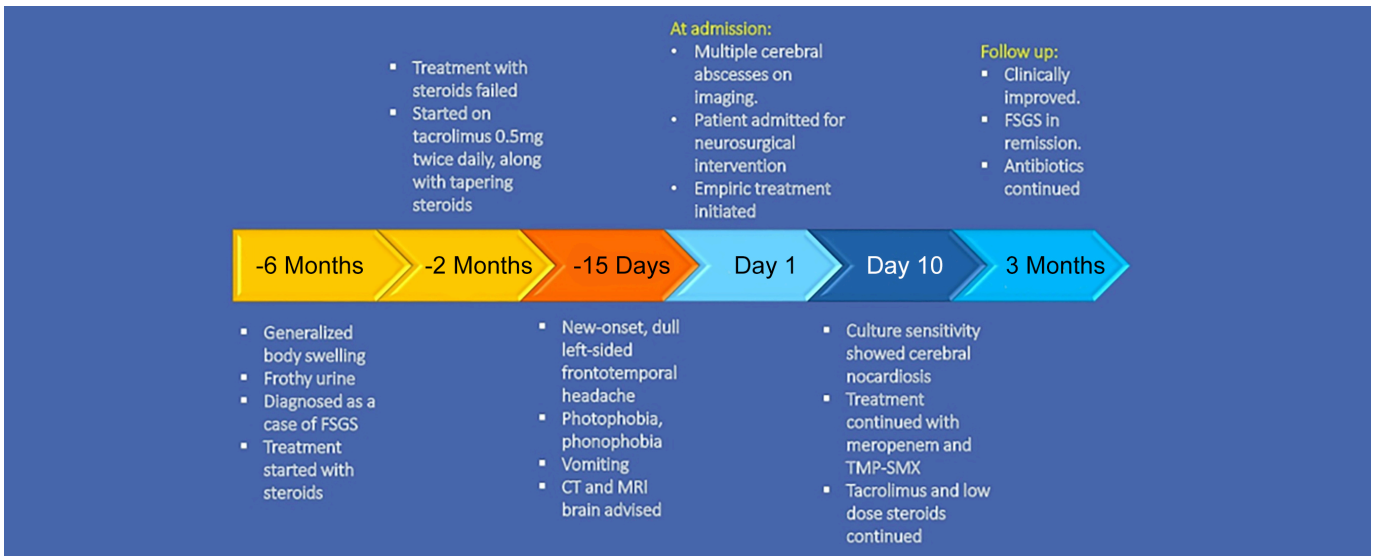


**Figure 1: Timeline of events (CT: Computed tomography; FSGS: Focal segmental glomerulosclerosis; MRI: Magnetic resonance imaging; TMP-SMX: Trimethoprim-sulfamethoxazole).**

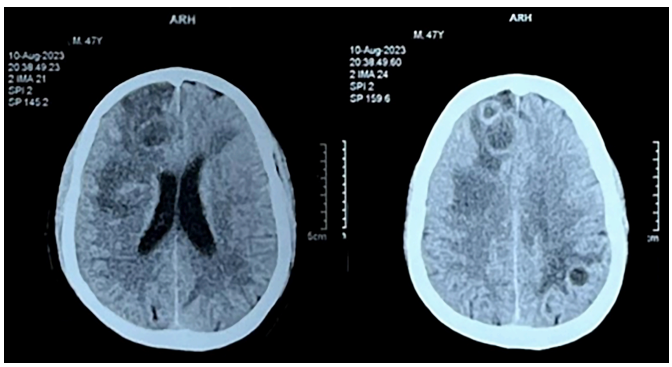


**Figure 2: CT scan of the brain showing multiple well-defined lesions suggestive of space-occupying lesions with surrounding vasogenic oedema.**

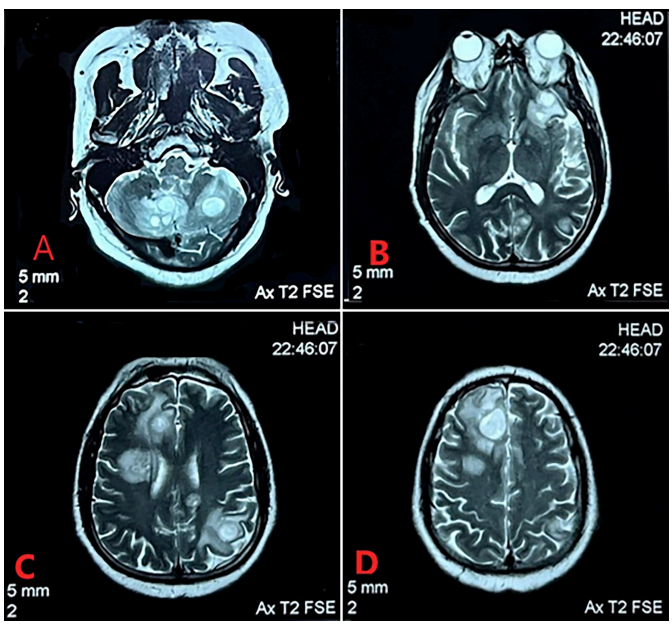


**Figure 3: MRI brain axial T2 slices showing hyperintense lesions with surrounding vasogenic oedema in (A) cerebellum, (B) left temporal grey matter, (C) right frontal (parasagittal), parietal and left occipital regions, and (D) right frontal region.**

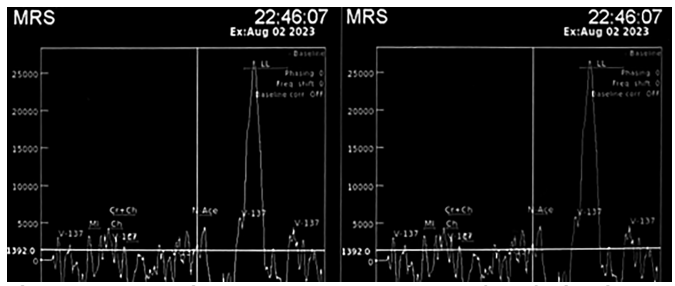


**Figure 4: Magnetic resonance spectroscopy (MRS) showing an elevated lipid lactate peak and decreased N-acetyl aspartate (NAA) signal within the core of the lesion.**

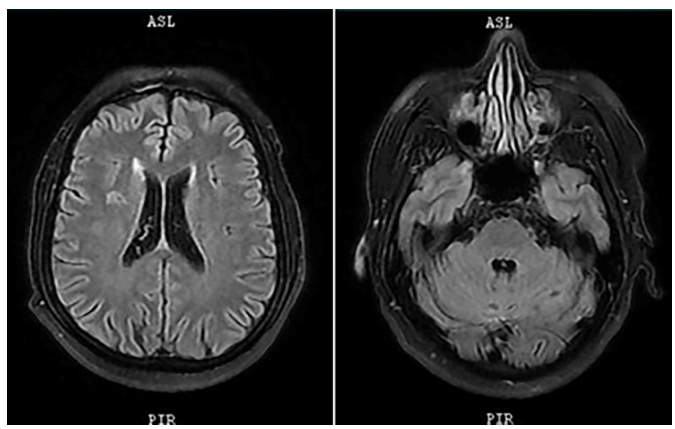


**Figure 5: MRI brain (T2 FLAIR) showing resolved cerebral abscess.**

His vitals at presentation were pulse of 85 bpm, blood pressure (BP) of 140/90 mmHg, $SpO_2$ of 98% at room air, temperature of 98.6°F, and a body mass index (BMI) of 32 kg/m$^2$. On general physical examination, he had facial puffiness and bilateral lower-limb pitting oedema. Neurological examination showed no focal neurological deficit, apart from bilateral papilloedema on fundoscopy. The cardiac, respiratory, and abdominal examinations were unremarkable. CT brain without intravenous (IV) contrast showed well-defined fluid-filled lesions, with a hyperdense rim and surrounding vaso-

genic oedema at the grey-white matter junction in the right frontal area, in the parasagittal location. Three other similar lesions were noted in the left frontal and parietal area without any mass effect or mid-line shift (Figure 2).

MRI brain without contrast was performed owing to deranged renal functions, which showed multiple T2 hyperintense lesions with surrounding vasogenic oedema visualised in both supra- and infra-tentorial regions (Figure 3). Magnetic resonance spectroscopy (MRS) showed an elevated lipid lactate peak and decreased N-acetyl aspartate (NAA) signal within the core of the lesion (Figure 4), likely suggestive of a brain abscess.

At this point, our main differential included tuberculomas or bacterial brain abscesses. Echocardiography and high-resolution CT scan of the chest were normal, which ruled out pulmonary tuberculosis and infective endocarditis. Based on MRI and MRS findings, the patient was shifted to the neurosurgical ward for drainage of cerebral abscess. The pus culture and sensitivity analysis ascertained a cerebral abscess due to N. farcinica, sensitive to meropenem, amikacin, and trimethoprim-sulfamethoxazole.

Hence, after liaison with neurosurgery and infectious disease (ID) departments, the patient was started on injectable meropenem 500 mg thrice daily (renal adjusted dose), which was later increased to 2g thrice daily after renal functions resolved, and oral trimethoprim / sulfamethoxazole (160 / 800 mg) 2 tablets orally thrice daily. The expected duration of treatment for cerebral nocardiosis was 12 months. Meanwhile, tacrolimus and low-dose steroids (7.5 mg once daily) were continued for FSGS, which is now in remission.

The patient improved symptomatically and is still under three monthly OPD follow-ups of the neurosurgery, ID, and nephrology departments. At the third month, the patient's headache completely resolved. MRI brain with contrast at sixth month showed complete resolution of cerebellar, occipital, and temporal abscesses. Altered signal intensity showing a resolved cerebral abscess in the right frontal lobe (Figure 5). He is continued on meropenem 2g IV thrice daily, trimethoprim / sulfamethoxazole (160 / 800 mg) twice daily, and tacrolimus 0.5mg twice daily.

## DISCUSSION

Cerebral nocardiosis mainly affects immunocompromised hosts owing to impaired T cell-mediated immunity.[6] T lymphocytes and lung macrophages locally control the spread of disease. Therefore, conditions and medications causing cellular immunodeficiency play a pivotal role in its development, which include patients on immunosuppressive medicines, solid organ transplant recipients, haematological malignancies, chronic obstructive pulmonary disease, HIV infection, and diabetes mellitus.[7,8] Anagnostou *et al*. labelled a person to be immunosuppressed if he is taking prednisolone or its equivalent at a dose of ≥10 mg daily for at least three months.[8] But it can also occur in immunocompetent hosts.[2] In Pakistan, until now, two cases have been reported of cerebral nocardiosis, one in an immunocompromised and the other in an immunocompetent host.[2] The common presenting features in cerebral nocardiosis include focal neurological deficits, fever, headache, and altered mental status in descending order. The focal deficits included seizures, motor weakness, and cranial nerve involvement.[9] Cerebral nocardiosis is diagnosed based on a CT, MRI of the brain with or without contrast, and identification of Nocardia species in cerebral abscess or cerebrospinal fluid analysis.[8]

This patient was started on steroids and tacrolimus (a calcineurin inhibitor) for FSGS, which led to immunosuppression. Later, he presented with a new onset headache without any focal neurological deficit. But when it did not respond to analgesics, an MRI of the brain was advised, which showed multiple ring-enhancing cerebral abscesses. The culture and sensitivity of these abscesses confirmed *Nocardia farcinica* growth. However, no other focus of infection was observed, which could have led to secondary cerebral involvement. So, it was labelled as primary cerebral nocardiosis. Minimal central nervous system symptoms without pulmonary or cutaneous involvement underlying primary cerebral nocardiosis led to this case report. The disease burden of cerebral nocardial infection in Pakistan is still unknown. With the invention of the latest technologies, culture and sensitivity, and a high index of suspicion, more cases of cerebral nocardiosis can be identified.

In central nervous system involvement, a multi-medicine regimen of antibiotics containing at least two medicines is preferred.[10] The combination includes co-trimoxazole with either imipenem, cefotaxime, or ceftriaxone, with or without amikacin. Linesolid can be used as part of a multi-medicine regime because of its broad activity against *Nocardia* species, but its combination with amikacin is usually avoided. The recommended doses of these medicines are trimethoprim 15 mg/kg/d, imipenem 2-3 g/d, amikacin 20-30 mg/kg/d, cefotaxime 6-12 g/d, ceftriaxone 2 g/d, and linezolid 1200 mg/d.[7] A total of 12 months of therapy is recommended in case of cerebral nocardiosis.[7] Apart from antibiotics, surgical excision should be considered for deep-seated cerebral abscesses.[7] The mortality of patients treated with antibiotics alone is 30%, as compared to 24 and 50% for abscess excision and aspiration, respectively.[4] The relapse rate of cerebral nocardiosis is around 13%, whether treated with antibiotics alone or combined with a neurosurgical approach; however, longer treatment >6 months reduces relapse rate.[8] This patient was treated with combined surgical aspiration and antibiotics. The main hurdles in treating this patient were the deranged renal profile, availability, and cost-effectiveness of prolonged IV medications.

In conclusion, with limited real-world data on cerebral nocardiosis in Pakistan, its true disease burden and epidemiology are unknown. The clinical significance of this case is paramount for both physicians and neurologists that nocardiosis can present without neurological deficit with raised intracerebral pressure and headache. In such cases, biopsy and culture / sensitivity give clues for diagnosis and antimicrobial therapy selection. Timely antibiotic initiation, close follow-up, and monitoring can halt disease progression and prevent complications of the disease.

**PATIENT'S CONSENT:**
Informed consent was obtained from the patients to publish the data concerning this case.

**COMPETING INTEREST:**
The authors declared no conflict of interest.

**AUTHORS' CONTRIBUTION:**
SA: Conception of the work, analysis, drafting, and accountability.
SR: Acquisition, analysis, and accountability.
IE: Analysis and final approval.
MA: Supervision.
RA: Editing and proofreading.
All authors approved the final version of the manuscript for publication.

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