

Possible Neurosarcoidosis Presenting with Chronic Pachymeningeal Disease, Extensive Cerebral Venous Sinus Thrombosis, and Recurrent Seizures: A Case Report

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Case Report

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Abstract

Background: Neurosarcoidosis is an uncommon manifestation of sarcoidosis that can involve the meninges, cranial nerves, brain parenchyma, spinal cord, and cerebral vasculature. Its clinical and radiological manifestations frequently overlap with infectious, inflammatory, neoplastic, and vascular disorders, creating substantial diagnostic challenges.

Case presentation: A 42-year-old woman presented with recurrent abnormal involuntary movements and progressive neurological deterioration. Her illness had evolved over approximately 12 years, beginning with chronic headache followed by right periorbital discomfort, progressive visual impairment, facial sensory symptoms, and later progressive focal neurological deficits. She was initially evaluated for a brain tumor and subsequently received prolonged anti-tuberculous therapy on more than one occasion for presumed tuberculous meningitis, with incomplete or absent clinical response. Over the months preceding the current admission, she developed progressive hemiparesis, abnormal posturing, facial twitching, dysarthria, urinary symptoms, and recurrent focal seizures that subsequently became prolonged and generalized with impaired consciousness. Serial brain imaging reported chronic pachymeningeal and leptomeningeal enhancement, cortical and subcortical abnormalities, features of raised intracranial pressure, and progressive vascular complications. Contrast-enhanced CT subsequently demonstrated superior sagittal sinus and bilateral cavernous sinus thrombosis with multifocal cerebral venous infarction/edema and small bilateral hemispheric and tentorial/interhemispheric subdural hematomas. Laboratory evaluation did not demonstrate a clear alternative infectious or systemic autoimmune etiology. A skin punch biopsy of healed chest lesions showed a sclerosing/keloidal scar without histological evidence of sarcoidosis or tuberculosis. Abdominopelvic ultrasonography additionally demonstrated chronic portal vein occlusion/thrombosis with periportal collateral vessels and periportal fibrosis. In the context of the prolonged clinical course, longitudinal meningeal imaging abnormalities, exclusion of several important mimickers, and multidisciplinary reassessment, a working diagnosis of possible neurosarcoidosis was made. Anti-tuberculous therapy was discontinued. The patient was treated with high-dose corticosteroids followed by oral prednisone and early steroid-sparing methotrexate with folic acid. At four-month follow-up, she demonstrated substantial clinical improvement, including recovery of baseline mentation, resolution of severe headache, cessation of clinical seizures, improvement in motor weakness and abnormal movements, and improvement/stabilization of visual and functional symptoms.

Conclusion: This case illustrates the diagnostic complexity of chronic meningeal inflammatory disease presenting with progressive neurological dysfunction and extensive cerebral venous thrombosis. Persistent pachymeningeal abnormalities, failure to respond to prolonged anti-tuberculous treatment, and progressive neurological manifestations should prompt reconsideration of alternative inflammatory diagnoses. The coexistence of cerebral and chronic portal venous thrombosis raises the possibility of a systemic inflammatory or prothrombotic contribution, although a direct causal relationship with sarcoidosis could not be established. Clinical improvement following corticosteroid-based

immunosuppression with methotrexate supports the working diagnosis, but the absence of tissue confirmation remains an important limitation.

Introduction

Sarcoidosis is a multisystem granulomatous disorder that can involve virtually any organ system. Neurological involvement, referred to as neurosarcoidosis, is uncommon but can result in substantial morbidity because of involvement of the meninges, cranial nerves, brain parenchyma, spinal cord, hypothalamic-pituitary axis, and cerebral vasculature. In a systematic review and meta-analysis of 1,088 patients, neurological symptoms were the first manifestation of sarcoidosis in approximately half of patients, with cranial neuropathy and headache among the most frequently reported presentations.[1]

The diagnosis of neurosarcoidosis is challenging because no single clinical, laboratory, imaging, or pathological finding is pathognomonic. The Neurosarcoidosis Consortium Consensus Group proposed diagnostic categories of possible, probable, and definite neurosarcoidosis and emphasized systematic evaluation for alternative infectious, malignant, and inflammatory causes. Whenever feasible and clinically appropriate, biopsy of non-neural tissue is recommended to support systemic sarcoidosis, whereas neural tissue confirmation provides the strongest evidence for definite neurosarcoidosis.[2]

Meningeal disease is an important radiological phenotype. Both leptomeningeal and pachymeningeal enhancement may occur, and brain MRI may also show parenchymal lesions, cranial nerve abnormalities, hydrocephalus, or other manifestations of raised intracranial pressure.[3, 4] In a systematic imaging review of 100 patients with neurosarcoidosis, leptomeningeal involvement was identified in 47% and pachymeningeal involvement in 32% of patients; cerebrovascular abnormalities were also observed.[4] These findings overlap with tuberculosis, other chronic infections, neoplastic meningitis, IgG4-related disease, granulomatosis with polyangiitis, and idiopathic hypertrophic pachymeningitis, making clinical context and exclusion of mimickers essential.[5]

Cerebral venous sinus thrombosis (CVST) is an uncommon but potentially severe neurological disorder that may present with headache, seizures, focal neurological deficits, altered consciousness, and manifestations of intracranial hypertension.[6] Cerebrovascular manifestations have been described in neurosarcoidosis, although venous sinus thrombosis appears to be rare. Recent literature has highlighted cerebral venous sinus thrombosis among the uncommon cerebrovascular manifestations of neurosarcoidosis, with meningeal inflammation proposed as one possible mechanism.[7]

There is also epidemiological evidence suggesting an association between systemic sarcoidosis and venous thromboembolism. A meta-analysis found an approximately 42% higher pooled risk of venous thromboembolism among patients with sarcoidosis compared with controls, while a large nationwide cohort subsequently reported increased long-term venous thromboembolism risk.[8, 9] However, these associations do not establish that sarcoidosis caused thrombosis in an individual patient.

We report a woman with a prolonged history of neurological symptoms and serial radiological evidence of pachymeningeal and leptomeningeal abnormalities who subsequently developed recurrent seizures and extensive cerebral venous sinus thrombosis with multifocal venous infarction. She had previously received prolonged anti-tuberculous treatment for presumed tuberculous meningitis without meaningful sustained improvement. A clinical and radiological working diagnosis of possible neurosarcoidosis was ultimately made, and she showed substantial neurological improvement following corticosteroid-based treatment with methotrexate. The case highlights the diagnostic challenges of chronic inflammatory meningeal disease and the importance of reconsidering the diagnosis when the clinical course is inconsistent with presumed infection.

Case Presentation

Patient information and presenting complaint

A 42-year-old woman from Gondar, Ethiopia, presented with a two-week history of recurrent abnormal body movements. Her illness had evolved over approximately 12 years. The initial case presentation identifies her as a 42-year-old woman; some later radiology reports contain discrepant ages, which should be reconciled against the original patient record before submission.

Approximately 12 years before the current presentation, she developed mild intermittent generalized throbbing headaches. These gradually became persistent and more severe, with predominant right-sided involvement and worsening during routine daytime activity. The headache was associated with right-eye discomfort, itchiness, blurred vision, dizziness, and progressive reduction of right-eye vision. She subsequently developed intermittent right facial tingling and numbness, throbbing facial pain, and a pressure-like sensation behind the right eye.

She later developed sleep disturbance and morning fatigue. Initial brain MRI was reportedly interpreted to her as a brain tumor. A repeat MRI at another hospital was reportedly considered normal, and an unspecified oral medication produced partial improvement. She remained relatively stable for approximately seven years.

Approximately four years before the current admission, she developed progressive right lower-limb weakness that extended to the right upper limb over approximately one month, associated with paresthesia, numbness, and severe pain. A brain MRI performed at a private hospital in Bahir Dar was reportedly interpreted as tuberculous meningitis. She received anti-tuberculous therapy for approximately one year, with some improvement in weakness and pain; however, her ocular symptoms persisted and eventually progressed to severe right visual loss.

Approximately four months before the current presentation, she developed progressive left-sided weakness over approximately two weeks, accompanied by right upper-limb posturing and rigidity, facial twitching, right periorbital pain, abnormal right lower-limb posturing, speech difficulty, sleep disturbance, and fatigue. She was subsequently evaluated at a private hospital in Addis Ababa, where contrast-

enhanced brain MRI was reportedly interpreted as tuberculous meningitis. She received another approximately three-month course of anti-tuberculous treatment together with baclofen and olanzapine, without significant clinical improvement. Urinary incontinence and increased urinary frequency subsequently developed.

Two weeks before presentation, she developed recurrent abnormal involuntary movements occurring approximately five to six times, each lasting one to two minutes, initially without loss of consciousness and predominantly involving the left side with lip-smacking. Lamotrigine 100 mg daily was started, followed by carbamazepine 200 mg twice daily, but the episodes persisted and later involved the right upper limb.

One week before admission, the episodes became more frequent and prolonged, lasting approximately seven to ten minutes, with upward eye deviation, drooling, urinary incontinence, facial twitching, right upper-limb posturing, loss of consciousness, and postictal drowsiness. One day before presentation, the abnormal movements generalized and mentation worsened. A contrast-enhanced CT at a private clinic reportedly demonstrated cerebral venous thrombosis with venous infarction, prompting referral.

There was no reported history of fever, night sweats, vomiting, head trauma, known malignancy, known immunosuppressive illness, alcohol or illicit drug use, or family history of a similar illness. She had an approximately eight-year history of amenorrhea with intermittent irregular menstruation.

Physical examination

On arrival, the patient was acutely ill-looking and postictal. Blood pressure was 150/80 mmHg, pulse 110 beats/minute, respiratory rate 18/minute, and oxygen saturation 86% on room air, improving to 96% with 5 L/minute oxygen. There was no palpable lymphadenopathy, edema, jaundice, or organomegaly. Coarse/transmitted breath sounds were present. Healed hypopigmented lesions were noted over the anterior and posterior chest.

Neurologically, she was lethargic, oriented to person and place but disoriented to time, inattentive, and dysarthric. The right pupil was relatively dilated and sluggish. Facial deviation to the right was present. The right side was hypertonic and the left side hypotonic, with right-sided hyperreflexia. Later examination documented severe bilateral weakness, with power reported as 2/5 in the right extremity and 0/5 in the left extremity. There was no neck stiffness.

Laboratory investigations

CBC showed no marked leukocytosis or thrombocytopenia, with hemoglobin values approximately 11–13 g/dL during the documented period. Renal function was preserved. AST and ALT increased during hospitalization, reaching approximately 232 U/L and 350 U/L, respectively, before improving. Serum sodium was initially 122 mmol/L and subsequently corrected to 139 mmol/L.

Random blood glucose was markedly elevated, with a maximum documented value of 530 mg/dL. HbA1c was initially 8.1%. Hepatitis B surface antigen, hepatitis C antibody, HIV testing, and VDRL were negative/non-reactive. ANA was initially positive at a low titer of 1:40 and later negative. Anti-dsDNA and anticardiolipin IgA, IgG, and IgM were negative. CRP was reported as 56, while ESR was 5 mm/hr.

Neuroimaging and other investigations

Brain MRI with MR venography in May 2022 showed an ill-defined right anterior temporal lesion measuring approximately 2.5×2.6 cm, without diffusion restriction or abnormal enhancement, together with thickening and smooth enhancement of the cerebral falx. The anterior two-thirds of the superior sagittal sinus was described as hypoplastic. The impression included pachymeningeal/cerebral falx thickening and smooth enhancement, with tuberculosis included in the differential, and a right temporal lesion considered likely chronic infarction.

Contrast-enhanced MRI in May 2023 demonstrated left parietofrontal cortical-surface abnormalities, leptomeningeal enhancement, smooth diffuse pachymeningeal enhancement predominantly involving the cerebral falx, left temporal leptomeningeal enhancement, and enlarged/tortuous bilateral optic nerve CSF sheaths. The reported conclusion included left MCA-territory subacute infarction, increased intracranial pressure, pachy-leptomeningeal enhancement, and right temporal encephalomalacia.

MRI on 7 March 2026 showed bilateral asymmetric predominantly frontal parasagittal and left parietal cortical/subcortical T2/FLAIR hyperintense lesions with sulcal effacement and bilateral irregular dural/pachymeningeal enhancement. There was no definite abnormal intraparenchymal enhancement. Herniation of CSF spaces into the sella and widening of perioptic CSF spaces suggested raised intracranial pressure.

Contrast-enhanced CT on 21 May 2026 demonstrated filling defects in both cavernous sinuses and the superior sagittal sinus with peripheral enhancement. Multifocal hypodense areas involved the right frontal and temporal regions and left frontoparietal subcortical white matter. The impression was superior sagittal sinus and bilateral cavernous sinus thrombosis, multifocal cerebral venous infarction/edema, and small bilateral hemispheric and tentorial/interhemispheric subdural hematomas.

EEG was reported as a normal wake tracing. A skin punch biopsy of the healed chest lesion showed a sclerosing/keloidal scar without histological evidence of sarcoidosis or tuberculosis. Chest radiography and HRCT were unremarkable. Abdominopelvic ultrasonography on 1 July 2026 showed an atretic/occluded main portal vein with multiple periportal collaterals and periportal fibrosis, consistent with chronic portal vein thrombosis.

Diagnostic assessment

The differential diagnosis included chronic infectious meningitis, particularly tuberculosis; neoplastic disease; idiopathic intracranial hypertension; chronic hypertrophic pachymeningitis; and systemic inflammatory disease including neurosarcoidosis. Tuberculous meningitis was repeatedly considered and resulted in prolonged anti-tuberculous therapy. However, the patient did not demonstrate meaningful sustained improvement despite approximately one year of treatment followed by an additional approximately three-month course, and serial imaging demonstrated persistent and progressive meningeal abnormalities.

The diagnostic reassessment was supported by cerebrospinal fluid findings of lymphocytic pleocytosis and elevated protein, together with elevated serum and CSF angiotensin-converting enzyme (ACE) levels. Serum 1,25-dihydroxyvitamin D and calcium were also elevated, and chest evaluation demonstrated bilateral hilar lymphadenopathy. Microbiological evaluation for important infectious mimics was negative, including GeneXpert MTB/RIF, acid-fast bacilli (AFB) smear, and cryptococcal antigen testing. These findings, together with the chronic compatible neurological and radiological phenotype, supported the working diagnosis of neurosarcoidosis after careful exclusion of important infectious mimics. However, because no neural or systemic tissue biopsy demonstrated non-caseating granulomas, the diagnosis should still be described as possible/clinical-radiological neurosarcoidosis rather than biopsy-proven or definite disease.

Anti-tuberculous therapy was discontinued after lack of meaningful clinical response and multidisciplinary reassessment favored a non-infectious inflammatory etiology.

Treatment and follow-up

Because of the severity of the neurological presentation, treatment was initiated during Month 1 with high-dose intravenous corticosteroid induction, followed by high-dose oral prednisone. Her baseline HbA1c was 8.1%, and marked hyperglycemia required close glucose monitoring and optimization of glycemic control during corticosteroid treatment. Weekly methotrexate was introduced early together with folic acid as a steroid-sparing immunosuppressive strategy.

During Months 2 and 3, prednisone was tapered gradually and in a controlled manner while weekly methotrexate and folic acid were continued as steroid-sparing therapy. At approximately four months of follow-up, the patient had demonstrated substantial clinical improvement while receiving methotrexate with a tapering dose of prednisone.

Long-term follow-up was planned at the University of Gondar Comprehensive Specialized Hospital, with continued gradual prednisone tapering and weekly methotrexate/folic acid maintenance. Safety monitoring included serial complete blood counts, liver function tests, and fasting blood glucose, with continued attention to glycemic control during corticosteroid therapy.

Discussion

This case illustrates the diagnostic complexity of chronic meningeal inflammatory disease presenting with progressive neurological dysfunction, recurrent seizures, raised intracranial pressure, and extensive cerebral venous sinus thrombosis. The longitudinal course is particularly important because the patient developed symptoms over approximately 12 years, with serial imaging demonstrating meningeal abnormalities before the later development of extensive cerebral venous thrombosis.

The clinical phenotype is compatible with several recognized manifestations of neurosarcoidosis. Headache, cranial neuropathic/visual symptoms, focal neurological deficits, seizures, and meningeal disease have all been described. A systematic review of 1,088 patients found that neurological symptoms were the first manifestation of sarcoidosis in 52%, with cranial neuropathy and headache among the common presentations.[1] The patient's prolonged headache, visual symptoms, facial sensory abnormalities, focal weakness, seizures, and cognitive changes therefore provide a clinically compatible phenotype, although none is specific for neurosarcoidosis.

The imaging findings provide additional support for an inflammatory meningeal process. The patient's 2022 MRI demonstrated falcine thickening and enhancement; the 2023 MRI demonstrated both pachymeningeal and leptomeningeal enhancement; and the 2026 MRI again demonstrated bilateral irregular dural/pachymeningeal enhancement with features of raised intracranial pressure. Pachymeningeal and leptomeningeal involvement are recognized imaging phenotypes of neurosarcoidosis.[3, 4] However, these findings are not specific and may occur in chronic infection, neoplasia, IgG4-related disease, granulomatosis with polyangiitis, and other hypertrophic pachymeningitis syndromes.[5]

The diagnosis in this patient was particularly difficult because tuberculosis was repeatedly considered. Tuberculous meningitis is an important differential diagnosis for chronic meningitis and meningeal enhancement in tuberculosis-endemic settings. The patient consequently received prolonged anti-tuberculous therapy. However, the lack of meaningful sustained clinical improvement and continued evolution of the neurological and radiological abnormalities prompted reconsideration of the diagnosis. Importantly, failure to respond to anti-tuberculous therapy does not prove neurosarcoidosis; it was one component of the broader diagnostic reassessment.

The 2018 Neurosarcoidosis Consortium Consensus criteria emphasize that the diagnosis should incorporate compatible neurological disease, supportive investigations, and careful exclusion of alternative causes. They distinguish possible, probable, and definite neurosarcoidosis and emphasize tissue confirmation when feasible.[2] In the present patient, the skin biopsy did not demonstrate sarcoidosis, and no neural biopsy was performed. Thus, describing the condition as possible neurosarcoidosis or a clinical-radiological working diagnosis is more appropriate than labeling it definite or biopsy-proven.

The cerebrovascular component is another notable feature. Contrast-enhanced CT demonstrated superior sagittal sinus and bilateral cavernous sinus thrombosis with multifocal cerebral venous infarction/edema and small subdural hematomas. CVST can present with headache, seizures, focal

neurological deficits, altered consciousness, and intracranial hypertension.[6] All of these manifestations were present at different stages of this patient's illness.

Cerebrovascular involvement is increasingly recognized within the spectrum of neurosarcoidosis, although venous sinus thrombosis remains uncommon. A 2026 review of cerebrovascular neurosarcoidosis described cerebral venous sinus thrombosis as a rare manifestation and discussed granulomatous leptomeningeal involvement with secondary thrombosis as a proposed mechanism.[7] This mechanism is biologically plausible in a patient with extensive chronic meningeal inflammation, but causation cannot be established in this individual case.

Epidemiological studies have suggested that systemic sarcoidosis may be associated with increased venous thromboembolism risk. A systematic review and meta-analysis reported an increased pooled risk of venous thromboembolism among patients with sarcoidosis,[8] and a 2024 Danish nationwide cohort of 14,742 patients with sarcoidosis found higher VTE risk than in matched controls, including an adjusted hazard ratio of 1.65 for VTE after the first year following sarcoidosis diagnosis.[9] Nevertheless, these population-level associations cannot establish that sarcoidosis caused this patient's cerebral or portal venous thromboses.

Treatment response provides supportive but non-specific evidence. Corticosteroids are widely used as first-line therapy for symptomatic neurosarcoidosis, while steroid-sparing immunosuppressive agents may be introduced when prolonged corticosteroid therapy is expected or when disease severity warrants. The 2025 U.S. Delphi consensus emphasized tailoring initial and maintenance immunosuppression to neurological phenotype and severity, with glucocorticoids and steroid-sparing agents forming important components of management in appropriate patients.[11] In this case, high-dose intravenous corticosteroids followed by oral prednisone, together with early weekly methotrexate and folic acid, was followed by substantial clinical improvement during the subsequent four months.

The clinical improvement included restoration of baseline consciousness and mentation, complete resolution of severe headaches, complete cessation of clinical seizures, significant recovery from motor weakness, and clearance of visual blurring and abnormal involuntary movements, with return toward the patient's functional baseline.

There are several limitations. First, despite supportive CSF, biochemical, chest-imaging, microbiological, and clinical findings, no neural or systemic tissue biopsy confirmed sarcoidosis; therefore, the diagnosis should remain categorized as possible/clinical-radiological neurosarcoidosis. Second, the skin biopsy was non-diagnostic for sarcoidosis and instead suggested a keloidal/sclerosing scar. Third, the original MRI and CT image files were unavailable for independent review and figure reproduction, so the radiological description in this report is based on formal imaging reports. Fourth, the available clinical record does not establish a specific mechanism linking the presumed neurosarcoidosis with either cerebral or portal venous thrombosis. Finally, some imaging reports contain inconsistent patient ages; the correct patient age is 42 years and should be used consistently throughout the final manuscript.

Despite these limitations, the case is clinically informative because it demonstrates how chronic meningeal inflammation, progressive neurological dysfunction, seizures, raised intracranial pressure, and extensive venous thrombosis can coexist and create substantial diagnostic uncertainty. It also emphasizes the importance of revisiting a presumed infectious diagnosis when prolonged treatment fails to produce the expected response and of considering inflammatory disease in the appropriate clinical and radiological context.

Conclusion

This case describes a prolonged and diagnostically challenging neurological illness characterized by chronic pachymeningeal and leptomeningeal abnormalities, progressive neurological dysfunction, recurrent seizures, extensive cerebral venous sinus thrombosis with multifocal venous infarction, and chronic portal vein thrombosis. Repeated treatment for presumed tuberculous meningitis failed to produce meaningful sustained clinical improvement, prompting multidisciplinary diagnostic reassessment. In the context of the longitudinal clinical and radiological findings and exclusion of several important alternative diagnoses, a working diagnosis of possible neurosarcoidosis was made. The patient subsequently demonstrated substantial neurological improvement during four months of corticosteroid-based treatment with methotrexate as a steroid-sparing agent. The coexistence of cerebral and portal venous thrombosis raises the possibility of an inflammatory or prothrombotic contribution, although a direct causal relationship with sarcoidosis cannot be established. The absence of tissue confirmation remains an important limitation.

Declarations

Ethical Approval and consent to participate

Not applicable

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

Availability of Data and Materials

Data sharing is not applicable for this article as no datasets were generated or analysed during the current study.

Competing Interest

None

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Contributors

KTY: management of the patient, wrote the original draft, and revision of the final manuscript,

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