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## Case Report

# Neurobrucellosis Manifesting as Secondary Hemiparkinsonism in a Veterinary Technician: An Occupational Health Case Report

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## Abstract

**Background:** Neurobrucellosis is a rare but serious complication of brucellosis, affecting 2-10% of systemic cases. Movement disorders, particularly parkinsonian features, represent an unusual manifestation that is poorly documented in medical literature. **Case Presentation:** We report a 51-year-old male veterinary technician who developed progressive neurological symptoms over two years, beginning with fine tremor in his right upper limb and evolving to secondary right hemiparkinsonism with functional impairment. The patient presented with systemic manifestations including fever, weight loss, arthralgia, and headaches. Neuroimaging revealed hyperintensity in the right inferior cerebellar peduncle with post-contrast enhancement. Laboratory investigations confirmed neurobrucellosis through positive Rose Bengal test, elevated Brucella antibodies in serum and cerebrospinal fluid (CSF), and positive PCR for Brucella DNA in CSF. The patient was treated with combination antibiotic therapy (doxycycline, rifampicin, and ceftriaxone) for six months, plus symptomatic treatment with levodopa/carbidopa. Clinical improvement was achieved with resolution of systemic symptoms and significant improvement in neurological manifestations, though mild residual parkinsonian features persisted at 24 month follow-up. **Conclusions:** This case highlights the importance of considering neurobrucellosis in the differential diagnosis of movement disorders, particularly in patients with occupational exposure to animals. Early diagnosis and appropriate treatment can lead to significant clinical improvement, though some neurological sequelae may persist. Enhanced occupational safety measures are crucial for preventing such complications in high-risk populations.

**Keywords:** neurobrucellosis; parkinson's disease; zoonotic disease; occupational exposure; movement disorders; veterinary medicine

## 1. Introduction

Neurobrucellosis represents a serious and uncommon complication of brucellosis, affecting approximately 2-10% of patients with systemic infection (Arazi et al., 2025; Zou et al., 2024). This zoonotic disease, caused by bacteria of the genus *Brucella*, continues to be a significant occupational health concern globally, affecting approximately half a million individuals annually (Yang et al., 2024). The neurological manifestations of brucellosis are diverse and often misleading, earning neurobrucellosis the reputation of being a "great imitator" in neurological diagnostics (Zhuang et al., 2024). While meningitis and meningoencephalitis represent the most common presentations, the association between neurobrucellosis and movement disorders, particularly parkinsonian features, remains insufficiently documented in medical literature (Fusetti et al., 2024).

Recent systematic reviews have identified only limited cases of neurobrucellosis-associated movement disorders, with parkinsonian features occurring in approximately 30.8% of reported movement disorder cases (Barad et al., 2022). Notably, 75% of these cases occurred in individuals with occupational exposure to animals or animal products, underscoring the critical importance of occupational safety measures (Haripriya et al., 2024).

This case report provides valuable insights into an unusual presentation of neurobrucellosis manifesting as secondary hemiparkinsonism in a healthcare professional with occupational exposure, contributing to the growing body of evidence suggesting potential connections between zoonotic infections and neurodegenerative processes.

## 2. Case Presentation

A 51-year-old male veterinary technician with over two decades of experience working with livestock in a rural region presented with progressive neurological symptoms developing over approximately two years. His occupational duties included routine animal examinations, vaccinations, and assistance with births, often performed without consistent use of personal protective equipment.

### 2.1. Medical History and Initial Presentation

The patient's medical history included grade 3 obesity, hyperopic astigmatism with presbyopia and keratoconus, and lumbar spondylodiscarthrosis. He maintained moderate physical activity with weekly participation in recreational sports. Clinical manifestations began in April 2018 with a fine, intermittent tremor in the right upper limb. By early 2019, the tremor had increased in frequency and intensity. In February 2019, he developed a constellation of systemic symptoms including intermittent fever, decreased appetite, arthralgia affecting large joints, significant weight loss (approximately 12 kg over three months), persistent headaches, nausea, rigors, and right testicular edema.

By late 2019, his condition had progressed to secondary right hemiparkinsonism, characterized by asymmetric tremor, bradykinesia, and rigidity affecting the right side of his body. The patient reported increasing difficulty with fine motor skills, particularly writing, and changes in his normal gait pattern.

### 2.2. Clinical Examination

Neurological examination revealed mild hemiparesis with diminished muscle strength in the right limbs. Deep tendon reflexes were symmetrically present but diminished (grade II/V bilaterally). The most prominent feature was a fine distal tremor affecting the right upper and lower limbs, more pronounced during intentional movements, significantly interfering with writing and fine manipulative activities.

### 2.3. Investigations

Standard laboratory investigations showed mild elevation in inflammatory markers. Erythrocyte sedimentation rate was elevated at 42 mm/h (reference range 0-20 mm/h), and C-reactive protein measured 3.8 mg/dL (reference range  $\leq 0.5$  mg/dL).

Magnetic resonance imaging (MRI) of the brain revealed focal hyperintensity in the anterior aspect of the right inferior cerebellar peduncle. Post-contrast images demonstrated discrete enhancement of the pial covering, consistent with an inflammatory or infectious process. Additional findings included thickening of the mucosa in multiple sinuses, suggestive of concurrent pansinusitis.

Serological testing confirmed the diagnosis of neurobrucellosis. The Rose Bengal test was positive in both serum and cerebrospinal fluid (CSF). Enzyme-linked immunosorbent assay (ELISA) demonstrated elevated IgG antibodies against *Brucella* antigens in both serum and CSF samples. The

serum agglutination test yielded a titer of 1:320. Cerebrospinal fluid analysis revealed lymphocytic pleocytosis (85% lymphocytes) with a total cell count of 120 cells/mm<sup>3</sup>. Protein levels were elevated at 112 mg/dL (reference range 15-45 mg/dL), while glucose was slightly decreased at 42 mg/dL, giving a CSF glucose ratio of 0.43. Polymerase chain reaction (PCR) for *Brucella* DNA in the CSF was positive, providing molecular confirmation of neuroinfection (Liu et al., 2025; Yu et al., 2023).

Dopamine transporter single-photon emission computed tomography (DaT-SPECT) showed asymmetric reduction in tracer binding in the right striatum, consistent with nigrostriatal dopaminergic deficit but without the typical pattern seen in idiopathic Parkinson's disease, supporting the diagnosis of secondary parkinsonism.

### 3. Differential Diagnosis

The patient's presentation posed a significant diagnostic challenge due to the evolving nature of symptoms and unusual combination of systemic and neurological manifestations. Initial considerations included essential tremor and early idiopathic Parkinson's disease. However, several atypical features raised concerns, including rapid progression, presence of systemic symptoms, and the patient's age.

The combination of neurological symptoms with systemic manifestations pointed toward an infectious or inflammatory process. Neurosarcoidosis, multiple sclerosis, and rheumatological disorders were considered but lacked supporting features (Shirazinia et al., 2024).

The patient's occupational history raised the possibility of zoonotic infections. Key considerations included:

1. Neurobrucellosis supported by occupational exposure, chronic progressive course, and combination of systemic and neurological symptoms
2. Neuroborreliosis (Lyme disease) considered due to potential tick exposures
3. Neurotuberculosis given its endemic nature and ability to cause chronic meningitis

The positive serological tests for *Brucella* in both serum and CSF, coupled with PCR confirmation, ultimately established neurobrucellosis as the definitive diagnosis (Alikhani et al., 2024).

### 4. Treatment

Upon confirmation of neurobrucellosis, a multidisciplinary treatment approach was initiated. The cornerstone was prolonged combination antibiotic therapy following established protocols (Fusetti et al., 2024):

- Doxycycline 100 mg orally twice daily
- Rifampicin 600 mg orally once daily
- Ceftriaxone 2 g intravenously once daily for the first 30 days

After the initial 30-day intravenous phase, treatment continued with oral doxycycline and rifampicin for a total duration of 6 months. Regular liver function monitoring was performed to detect potential hepatotoxicity.

For parkinsonian symptoms, levodopa/carbidopa (100/25 mg) was initiated at half a tablet three times daily, gradually titrated based on symptomatic response. The patient demonstrated modest improvement in tremor and bradykinesia. Non-pharmacological interventions included physical therapy with emphasis on gait training and balance exercises, and occupational therapy to address functional impairments.



## 5. Outcome and Follow-Up

The patient was followed for 24 months after treatment initiation. Systemic symptoms resolved completely by the third month of treatment. Follow-up serological testing showed progressive decline in Brucella antibody titers, from initial 1:320 to 1:80 by the end of antibiotic course.

Neurological manifestations showed more gradual improvement. The tremor amplitude decreased considerably, though mild residual fine tremor persisted. Bradykinesia and rigidity showed substantial improvement, with the patient regaining approximately 80% of previous motor function by 12 months. The mild hemiparesis resolved completely by 9 months (Naderi et al., 2022).

Cerebrospinal fluid analysis normalized completely by 6 months, with negative PCR for Brucella DNA in all follow-up samples. Unified Parkinson's Disease Rating Scale (UPDRS) motor score improved from 28 to 12 by 24-month follow-up.

Repeat brain MRI at 12 months showed complete resolution of the previously noted hyperintensity and no evidence of active inflammation. The patient returned to full-time work with enhanced protective measures by 18 months.

## 6. Discussion

This case represents a rare manifestation of neurobrucellosis presenting as secondary hemiparkinsonism in an occupationally exposed healthcare worker. Neurobrucellosis affects only 3-5% of patients with systemic brucellosis, and movement disorders represent an even rarer presentation (Razaghi et al., 2025).

### 6.1. Pathophysiological Mechanisms

The pathophysiology of neurobrucellosis involves direct bacterial invasion of the central nervous system and immune-mediated inflammatory responses (He et al., 2022). The development of parkinsonian features likely resulted from inflammatory processes affecting the nigrostriatal dopaminergic pathway. Brucella-induced inflammation can lead to microglial activation and subsequent release of pro-inflammatory cytokines, potentially causing degeneration of dopaminergic neurons in the substantia nigra. The cerebellar involvement observed in our patient's MRI findings is consistent with the known propensity of Brucella to cause granulomatous inflammation in various CNS locations (Zhang et al., 2024).

### 6.2. Diagnostic Considerations

The diagnosis of neurobrucellosis requires high clinical suspicion, particularly in endemic regions or patients with relevant occupational exposures. Current WHO guidelines recommend diagnosis based on clinical features, evidence of CNS inflammation, positive serology or culture, and response to specific treatment (Salcedo et al., 2024).

The use of PCR for Brucella DNA in CSF has emerged as a valuable diagnostic tool, with sensitivity and specificity exceeding 90% in recent studies (Mongkolrattanothai et al., 2017; Fan et al., 2018). This approach is particularly valuable in culture-negative cases or when prior antibiotic use may have compromised bacterial viability.

Functional neuroimaging, particularly DaT-SPECT, proved valuable in our case by demonstrating dopaminergic dysfunction consistent with secondary parkinsonism, helping differentiate between primary neurodegenerative and secondary inflammatory causes.

### 6.3. Therapeutic Approaches

Treatment of neurobrucellosis with parkinsonian features requires addressing both the infectious process and movement disorder. Current consensus recommends prolonged combination antibiotic therapy with agents achieving adequate CNS penetration (Erdem et al., 2012).

A systematic review analyzing outcomes in 215 neurobrucellosis cases found that tripleantibiotic regimens including a third-generation cephalosporin were associated with better neurological outcomes compared to dual therapy (87% vs. 73% complete recovery) (Fusetti et al., 2024).

The optimal duration of therapy remains controversial. While most guidelines recommend 3-6 months of treatment, cases with focal neurological deficits may justify longer treatment courses (Alhatou et al., 2024).

6.4. Occupational Health Implications

This case underscores the critical importance of occupational health measures in preventing zoonotic infections among high-risk populations. The CDC has published comprehensive guidelines for brucellosis prevention in occupational settings, recommending appropriate personal protective equipment, engineering controls, regular health surveillance, and education on safe work practices (Powers et al., 2020).

The potential severity of neurological complications from occupationally-acquired brucellosis reinforces the need for stringent adherence to preventive measures, particularly in resource-limited settings where access to protective equipment may be constrained.

6.5. Comparison with Published Cases

Table 1 summarizes published cases of neurobrucellosis-associated movement disorders, highlighting similarities and differences compared to our patient.

Table 1. Published cases of neurobrucellosis with movement disorders.

Author (Year)	Age/Sex	Presenting Features	Occupation	Outcom
Barad et al. (2022)	65/M	Bilateral parkinsonism, fever	Farmer	Partial r
Yasin and Mojdehi (2014)	78/M	Acute parkinsonism, SIADH	Unknown	Good re
Kim et al. (2015)	60/M	Aphasia, weakness	Unknown	Significa
Current case	51/M	Right hemiparkinsonism	Veterinary technician	Good re

A systematic review identified 26 cases of neurobrucellosis with movement disorders between 1980 and 2020, with only 8 cases (30.8%) presenting with parkinsonian features. Notably, 6 of these 8 cases (75%) occurred in individuals with occupational exposure to animals or animal products (Yang et al., 2024).

6.6. Limitations and Future Directions

Several limitations should be acknowledged. The causal relationship between brucellosis and parkinsonism, while strongly suggested by temporal sequence and clinical improvement, cannot be definitively proven. The possibility of coincidental idiopathic Parkinson’s disease cannot be completely excluded, though the atypical DaT-SPECT pattern argues against this.

Future research should focus on elucidating precise mechanisms by which Brucella infection leads to dopaminergic dysfunction and developing more targeted therapeutic strategies. Longitudinal studies of occupationally-exposed populations would provide valuable data on incidence and natural history of neurological complications.

7. Learning Points

- Neurobrucellosis should be considered in the differential diagnosis of movement disorders, particularly secondary parkinsonism, especially in patients with occupational animal exposure
- Neurological manifestations can be the predominant presentation, with CSF analysis, serology, and PCR being essential diagnostic components
- Effective management requires dual approach: prolonged combination antibiotic therapy (minimum 6 months) and symptomatic treatment with dopaminergic agents

- Appropriate personal protective equipment and occupational safety protocols are critical preventive measures for veterinary professionals
- While treatment can lead to significant improvement, residual neurological deficits may persist, highlighting the importance of early diagnosis and intervention

## 8. Patient Perspective

The patient provided the following perspective on his experience:

“When the tremor first started, I thought it was just fatigue from work. As a veterinary technician with over twenty years of experience, I was used to physical strain. But when it persisted and affected my ability to work especially drawing blood from animals I knew something was wrong. The most frightening part was how rapidly my condition deteriorated with fever and weight loss. Multiple doctors suggested everything from stress to early Parkinson’s disease. The diagnosis of neurobrucellosis was both shocking and relieving because I thought I was being careful at work, and relieving because finally there was an explanation and treatment. I realized I had become complacent about safety measures, sometimes skipping gloves for quick procedures and consuming local dairy products. The treatment was challenging, but physical therapy helped tremendously. Returning to work was anxiety-provoking, but I’m now much more vigilant about protective equipment and have become a safety advocate among my colleagues. While I still have mild tremor when tired, I’ve learned to adapt. The experience changed how I view my profession and health, with a much deeper respect for the invisible risks we face when working with animals.”

## 9. Conclusions

This case report documents a rare presentation of neurobrucellosis manifesting as secondary hemiparkinsonism in a veterinary technician. The case highlights the importance of maintaining high clinical suspicion for neurobrucellosis in patients with neurological symptoms and occupational animal exposure, particularly in endemic regions.

Early diagnosis through appropriate serological and molecular testing, combined with prolonged combination antibiotic therapy and symptomatic management, can lead to significant clinical improvement. However, some neurological sequelae may persist, emphasizing the critical importance of prevention through enhanced occupational safety measures.

This case contributes to the limited literature on movement disorders associated with neurobrucellosis and underscores the need for continued research into the mechanisms underlying this rare but serious complication of brucellosis.

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