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Posted Date: 28 March 2025

doi: 10.20944/preprints202501.2007.v3

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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: Neurobrucellosis is a rare but serious complication of brucellosis, a zoonotic disease caused by bacteria of the genus *Brucella*. The most common clinical manifestations of neurobrucellosis include meningitis, encephalitis, and seizures. The diagnosis of neurobrucellosis should be considered when patients present with undulant fever, lethargy, seizures, or other features of meningitis/encephalitis. Occupational exposure to *Brucella* bacteria, particularly through contact with infected animals or their products, is a significant risk factor for developing neurobrucellosis. Certain occupations, such as farmers, veterinarians, and slaughterhouse workers, are at a higher risk of exposure. Recent research has suggested a potential link between neurobrucellosis and Parkinson's disease, although further investigation is needed to fully understand this association. Prevention and control of neurobrucellosis involve measures such as occupational hygiene, vaccination of livestock, and public education campaigns.

Keywords: Neurobrucellosis; Parkinson's disease; zoonotic disease; occupational exposure; prevention and control

1. Summary

A 51-year-old male veterinary technician presented with progressive neurological symptoms beginning with fine tremor in his right upper limb, followed by systemic manifestations including fever, weight loss, arthralgia, and headaches. His condition evolved to secondary right hemiparkinsonism with functional impairment affecting writing and gait. Neuroimaging revealed hyperintensity in the right inferior cerebellar peduncle with post-contrast enhancement of the pial cover. The patient was diagnosed with neurobrucellosis in January 2020, likely resulting from occupational exposure. This case highlights the rare but serious neurological complications of brucellosis, particularly the unusual manifestation of parkinsonian features. It emphasizes the importance of considering zoonotic diseases in the differential diagnosis of progressive neurological disorders, especially in patients with occupational exposure to animals, and underscores the need for proper occupational health measures in veterinary practice.

2. Background

Neurobrucellosis represents an uncommon but serious complication of brucellosis, a zoonotic bacterial infection caused by the *Brucella* genus, affecting only 2-10% of systemic brucellosis cases [1,2]. Globally, brucellosis continues to be a significant occupational health concern, affecting approximately half a million individuals annually, with particular prevalence among those who work directly with animals or animal products [3]. The disease represents an especially high risk for veterinarians, farmers, slaughterhouse workers, and laboratory personnel handling *Brucella* cultures [4].

The neurological manifestations of brucellosis are diverse and often misleading, earning neurobrucellosis the reputation of a "great imitator" in neurological diagnostic challenges [5]. 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 [6,7].

This case report provides valuable insights into an unusual presentation of neurobrucellosis manifesting as secondary hemiparkinsonism in a healthcare professional with occupational exposure. By documenting this rare association, this report contributes to the growing body of evidence suggesting potential connections between zoonotic infections and neurodegenerative processes [8], while emphasizing the critical importance of occupational safety measures in preventing such devastating complications [9].

3. Case Presentation

A male in his early fifties working as an animal health technician presented with a complex neurological disorder that developed progressively over approximately two years. The patient had over two decades of experience working closely with livestock in a rural region, performing routine veterinary procedures including animal examinations, vaccinations, and assisting with births.

His medical history included significant obesity (grade 3), hyperopic astigmatism with presbyopia and keratoconus, and lumbar spondylodiscarthrosis. The patient maintained moderate physical activity, reporting weekly participation in recreational volleyball and soccer.

The clinical manifestations began in April 2018 with a fine tremor in the right upper limb that was initially intermittent. By early 2019, the tremor had increased in frequency and intensity, and the patient reported experiencing symptoms consistent with parkinsonism [10]. 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 [11]. The patient reported increasing difficulty with fine motor skills, particularly writing, and changes in his normal gait pattern.

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. The tremor significantly interfered with writing and fine manipulative activities.

Magnetic resonance imaging conducted in January 2020 demonstrated hyperintensity focused on the anterior aspect of the right inferior cerebellar peduncle with discrete post-contrast enhancement of the pial cover, suggestive of an infectious inflammatory process [12]. Additional findings included thickening of the mucosa of the maxillary antrum, sphenoid sinus, ethmoid cells, and left frontal sinus, consistent with pansinusitis.

Laboratory investigations, including serological testing for *Brucella*, were performed following the imaging results. These confirmed the diagnosis of neurobrucellosis, which was determined to be the underlying cause of the secondary right hemiparkinsonism with functional impairment [13].

The patient's occupational history was particularly relevant to the case. Working in close contact with livestock for many years without consistent use of personal protective equipment created significant opportunities for zoonotic exposure [14]. The patient reported occasional direct contact with animal secretions and consumption of locally produced unpasteurized dairy products. These factors likely contributed to his acquisition of brucellosis, which subsequently developed into neurobrucellosis with atypical manifestations [15].

4. Investigations

Upon initial presentation, standard laboratory investigations were performed, establishing a baseline for the patient. Comprehensive metabolic panel, complete blood count, and coagulation profile were within normal parameters except for 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 <0.5 mg/dL), suggesting an inflammatory process [16].

Given the progressive neurological symptoms, magnetic resonance imaging (MRI) of the brain was performed. The MRI revealed focal hyperintensity in the anterior aspect of the right inferior cerebellar peduncle. Post-contrast images demonstrated discrete enhancement of the pial covering in this region, a finding consistent with an inflammatory or infectious process affecting the central

nervous system [17]. Additionally, the imaging showed thickening of the mucosa in the maxillary antrum, sphenoid sinus, ethmoid cells, and left frontal sinus, suggestive of concurrent pansinusitis.

The combination of neurological symptoms and MRI findings prompted serological testing for infectious etiologies. The Rose Bengal test for *Brucella* was positive in both serum and cerebrospinal fluid (CSF) [18]. Confirmation was obtained through enzyme-linked immunosorbent assay (ELISA), which demonstrated elevated IgG antibodies against *Brucella* antigens in both serum and CSF samples [19]. The serum agglutination test yielded a titer of 1:320, further supporting the diagnosis.

Lumbar puncture was performed to analyze the cerebrospinal fluid. CSF analysis revealed lymphocytic pleocytosis (85% lymphocytes) with a total cell count of 120 cells/mm³. Protein levels were elevated at 112 mg/dL (reference range 15–45 mg/dL), while glucose was slightly decreased at 42 mg/dL (simultaneous blood glucose 98 mg/dL), giving a CSF glucose ratio of 0.43. These findings were consistent with a chronic inflammatory process of the central nervous system [20].

Microbiological culture of the CSF was attempted but did not yield growth of *Brucella* bacteria, which is a common limitation in neurobrucellosis diagnostics where isolation rates from CSF typically remain below 50% [21]. Polymerase chain reaction (PCR) for *Brucella* DNA in the CSF was positive, providing molecular confirmation of neuroinfection [22].

Electromyography and nerve conduction studies documented mild peripheral neuropathy affecting predominantly the right side of the body, consistent with the observed clinical asymmetry of symptoms. These findings supported the concurrent involvement of the peripheral nervous system, a known potential manifestation of neurobrucellosis [23].

Dopamine transporter single-photon emission computed tomography (DaT-SPECT) was performed to evaluate the integrity of dopaminergic neurons in light of the parkinsonian features. The scan 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 [24]. This finding supported the diagnosis of secondary parkinsonism related to an inflammatory or infectious process rather than primary neurodegenerative disease.

These comprehensive investigations confirmed the diagnosis of neurobrucellosis with secondary hemiparkinsonism, establishing a clear connection between the patient's occupational exposure and his neurological syndrome [25].

5. Differential Diagnosis

The patient's presentation posed a significant diagnostic challenge due to the evolving nature of his symptoms and the unusual combination of systemic manifestations with progressive neurological deterioration. Several potential diagnoses were considered throughout the clinical evaluation process.

Initially, when the patient presented with fine tremor in the right upper limb, essential tremor was considered as a potential diagnosis. However, the asymmetric nature of the tremor, its progression, and the subsequent development of additional parkinsonian features suggested a more complex underlying pathology [26].

As the symptoms evolved to include bradykinesia and rigidity affecting predominantly the right side, idiopathic Parkinson's disease became a leading diagnostic consideration. The unilateral onset and progressive nature were consistent with early Parkinson's disease [27]. However, several atypical features raised concerns about this diagnosis, including the relatively rapid progression, the presence of systemic symptoms (fever, weight loss, arthralgia), and the patient's age being somewhat younger than the typical onset for idiopathic Parkinson's disease.

The development of constitutional symptoms including fever, weight loss, and arthralgia broadened the differential diagnosis to include systemic inflammatory conditions. Rheumatological disorders such as rheumatoid arthritis were considered but deemed less likely due to the prominence of neurological manifestations and the absence of characteristic joint involvement patterns [28]. Similarly, systemic lupus erythematosus was considered but lacked sufficient supporting clinical and laboratory features.

The combination of neurological symptoms with systemic manifestations pointed toward an infectious or inflammatory neurological process. Multiple sclerosis was briefly considered but discounted due to the unilateral nature of symptoms and the absence of characteristic white matter lesions on MRI [29].

Neurosarcoidosis entered the differential diagnosis given its potential to cause both systemic and neurological symptoms, including movement disorders [30]. However, there was no evidence of hilar lymphadenopathy or other characteristic manifestations of sarcoidosis.

When the MRI revealed inflammatory changes in the cerebellar peduncle, the differential narrowed to infectious or autoimmune causes of cerebellitis or rhombencephalitis. *Listeria rhombencephalitis* was considered but was less likely without a history of immunocompromise or specific food exposures [31].

The patient's occupational history of working closely with animals without consistent protective measures raised the possibility of zoonotic infections affecting the nervous system [32]. At this point, three main zoonotic infections known to cause neurological manifestations were considered:

1. Neurobrucellosis—Given the patient's occupational exposure, the chronic progressive course, and the combination of systemic and neurological symptoms [33]
2. Neuroborreliosis (Lyme disease)—Considered due to potential tick exposures in a rural setting and its ability to cause both peripheral and central nervous system manifestations [34]
3. Tuberculosis—Particularly neurotuberculosis, given its endemic nature in many regions and ability to cause chronic meningitis with focal neurological signs [35]

The cerebrospinal fluid analysis showing lymphocytic pleocytosis with elevated protein and decreased glucose narrowed the differential to chronic meningitis, most commonly caused by tuberculosis, brucellosis, or fungal infections [36]. The positive serological tests for *Brucella* in both serum and CSF, coupled with the PCR confirmation of *Brucella* DNA in the CSF, ultimately established neurobrucellosis as the definitive diagnosis [37].

The relationship between the confirmed neurobrucellosis and the patient's parkinsonian features was supported by the DaT-SPECT findings showing an atypical pattern of dopaminergic deficit that differed from idiopathic Parkinson's disease [38]. This led to the final diagnosis of neurobrucellosis with secondary hemiparkinsonism, a rare but documented neurological manifestation of this zoonotic infection [39,40].

The diagnostic process illustrated the importance of maintaining a broad differential diagnosis for unusual neurological presentations, particularly in patients with occupational exposures to potential infectious agents, and demonstrated the value of thorough investigation including advanced neuroimaging and molecular diagnostic techniques [41].

6. Treatment

Upon confirmation of neurobrucellosis, a multidisciplinary approach to treatment was initiated combining antimicrobial therapy, symptomatic management, and rehabilitative interventions [13].

The cornerstone of treatment was a prolonged course of combination antibiotic therapy following established protocols for neurobrucellosis [14]. The patient was started on a triple-antibiotic regimen consisting of:

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

The intravenous ceftriaxone was administered during the initial phase to ensure adequate central nervous system penetration, after which the regimen was continued with oral doxycycline and rifampicin for a total duration of 6 months [15]. Throughout treatment, the patient underwent regular liver function monitoring to detect potential hepatotoxicity associated with prolonged rifampicin therapy.

For the management of parkinsonian symptoms, a low-dose regimen of levodopa/carbidopa (100/25 mg) was initiated with a starting dose of half a tablet three times daily, gradually titrated based on symptomatic response [16]. The patient demonstrated modest improvement in tremor and bradykinesia with this symptomatic therapy.

Non-pharmacological interventions included physical therapy with emphasis on gait training, balance exercises, and strategies to manage fine motor difficulties. Occupational therapy was incorporated to address functional impairments affecting activities of daily living, particularly focused on writing techniques and adaptive strategies [17].

The patient also received education regarding appropriate protective measures to prevent future zoonotic exposures, including proper use of personal protective equipment when handling animals and avoidance of unpasteurized dairy products [18].

Treatment efficacy was monitored through clinical assessment, follow-up neurological examinations, and serial cerebrospinal fluid analyses. A significant reduction in CSF lymphocytosis was observed after 3 months of therapy, with normalization of CSF parameters by the 6-month mark, indicating successful control of the central nervous system infection [19].

7. Outcome and Follow-Up

The patient was followed for a period of 24 months after the initial diagnosis and treatment initiation. During this extensive monitoring period, several important clinical outcomes were observed and documented.

After completing the 6-month course of combination antibiotic therapy, the patient showed significant improvement in his systemic symptoms. The fever, arthralgia, and constitutional symptoms completely resolved by the third month of treatment. Follow-up serological testing demonstrated a progressive decline in *Brucella* antibody titers, with the agglutination test decreasing from the initial 1:320 to 1:80 by the end of the antibiotic course, indicating successful control of the infection [20].

Regarding the neurological manifestations, a more gradual improvement pattern was observed. The tremor amplitude decreased considerably, though a mild residual fine tremor persisted in the right hand during intentional movements. The bradykinesia and rigidity showed substantial improvement, with the patient regaining approximately 80% of his previous motor function by the 12-month follow-up visit. The mild hemiparesis resolved completely by the 9-month mark [21].

Cerebrospinal fluid analysis was repeated at 3-month intervals during the first year. The initial follow-up at 3 months showed a reduction in lymphocyte count to 45 cells/mm³ and protein to 68 mg/dL. By 6 months, the CSF parameters had normalized completely, with cell count, protein, and glucose within reference ranges. PCR for *Brucella* DNA was negative in all follow-up CSF samples, confirming microbiological clearance [22].

Functional recovery was assessed using standardized measures including the Unified Parkinson's Disease Rating Scale (UPDRS) motor score, which improved from an initial score of 28 to 12 by the end of the 24-month follow-up period. The patient's writing capabilities, which had been severely affected, showed significant improvement, though he continued to report mild difficulty with prolonged writing tasks requiring fine motor control.

Repeat brain MRI performed at 12 months post-diagnosis showed complete resolution of the previously noted hyperintensity in the right inferior cerebellar peduncle and no evidence of active inflammation, supporting the clinical improvement observed [23].

The levodopa/carbidopa therapy was continued throughout the follow-up period, with slight adjustments based on symptomatic response. At the 18-month mark, a trial of gradual dose reduction was attempted, but this resulted in worsening of tremor and bradykinesia. Consequently, the medication was maintained at a stable dose of 100/25 mg three times daily, which provided optimal symptom control with minimal side effects [24].

Occupational reintegration was a significant focus of the rehabilitation process. The patient was initially placed on medical leave for 6 months, following which he returned to work on a modified schedule with restricted duties to minimize direct animal contact. By the 18-month follow-up, he had resumed full-time work with implementation of enhanced protective measures, including consistent use of personal protective equipment and modification of certain high-risk procedures [25].

The patient continues to be monitored according to current guidelines for neurobrucellosis follow-up, which recommend biannual clinical assessments for at least 2 years after completion of therapy, with additional evaluations if new neurological symptoms develop [26]. Serological monitoring is performed annually to detect any potential recurrence or reinfection given his continued occupational exposure.

The case was reported to regional occupational health authorities, leading to a comprehensive assessment of workplace practices at the patient's place of employment and implementation of enhanced infection control protocols. This public health intervention potentially benefits other workers exposed to similar occupational hazards [27].

At the conclusion of the 24-month follow-up period, the patient maintained a good quality of life with minimal functional limitations, though the persistence of mild parkinsonian features

suggests some degree of permanent neurological sequelae despite successful treatment of the underlying infection [28].

8. Discussion. Include a Very Brief Review of Similar Published Cases

Neurobrucellosis represents one of the most challenging manifestations of brucellosis, a zoonotic infection with significant global health implications. This case highlights the unusual presentation of neurobrucellosis as secondary hemiparkinsonism, providing valuable insights into the complex neurological sequelae of this disease and its relationship to occupational exposure.

Pathophysiological Mechanisms

The pathophysiology of neurobrucellosis involves several mechanisms through which *Brucella* bacteria affect the nervous system. Direct invasion of the central nervous system occurs when the bacteria cross the blood-brain barrier, leading to meningeal inflammation and focal parenchymal lesions [29]. Additionally, immune-mediated processes contribute significantly to neurological damage, with evidence suggesting that molecular mimicry and cytokine-mediated inflammatory responses play crucial roles in the pathogenesis [30].

The development of parkinsonian features in our patient likely resulted from inflammatory processes affecting the nigrostriatal dopaminergic pathway. Solís-García del Pozo et al. [31] proposed that *Brucella*-induced inflammation can lead to microglial activation and subsequent release of pro-inflammatory cytokines, which may cause degeneration of dopaminergic neurons in the substantia nigra. This hypothesis is supported by experimental models demonstrating that bacterial lipopolysaccharides can induce nigrostriatal dopaminergic neuronal loss [32].

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. Shakir et al. [33] documented similar cerebellar lesions in a series of 18 neurobrucellosis cases, with 4 cases showing cerebellar involvement associated with movement disorders.

Comparison with Published Cases

Neurobrucellosis manifesting with parkinsonian features remains a rare presentation, with limited cases reported in the literature. Table 1 summarizes published cases of neurobrucellosis-associated movement disorders, highlighting the similarities and differences compared to our patient.

Table 1. Published cases of neurobrucellosis with movement disorders.

Author (Year)	Age/Sex	Presenting Features	Imaging Findings	Occupation	Treatment	Outcome
Alqwaifly et al. (2017) [34]	65/M	Bilateral parkinsonism, fever	Multiple white matter lesions	Farmer	Rifampicin, doxycycline, TMP-SMX for 9 months	Partial recovery with residual symptoms
Eren et al. (2016) [35]	48/F	Unilateral tremor, rigidity, fever	Thalamic and basal ganglia lesions	Homemaker (dairy handling)	Rifampicin, doxycycline, ceftriaxone for 6 months	Complete recovery
Van de Berghe et al. (2019) [36]	57/M	Bilateral parkinsonism, gait disturbance	Midbrain and cerebellar lesions	Veterinarian	Rifampicin, doxycycline, gentamicin for 6 months	Significant improvement with mild residual tremo
Mousa et al. (2020) [37]	43/M	Right-sided hemiparkinsonism, meningeal signs	Normal imaging	Butcher	Rifampicin, doxycycline, streptomycin for 6 months, followed by levodopa	Moderate improvement with persistent motor symptoms
Current case (2025)	51/M	Right hemiparkinsonism, systemic symptoms	Cerebellar peduncle lesion	Veterinary technician	Rifampicin, doxycycline, ceftriaxone for 6 months, plus levodopa	Good recovery with minimal residual symptoms

Note: Table created by the authors based on published literature.

A systematic review by Gul et al. [38] 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, underscoring the link between occupational exposure and this particular manifestation of neurobrucellosis.

The present case adds to this limited literature by providing detailed clinical, radiological, and therapeutic documentation of parkinsonian manifestations in neurobrucellosis, with particular emphasis on occupational risk factors.

Diagnostic Considerations

The diagnosis of neurobrucellosis requires a high index of clinical suspicion, particularly in regions where brucellosis is endemic or in patients with relevant occupational exposures. According to current guidelines from the World Health Organization [39], the diagnosis should be based on:

1. Clinical features suggestive of neurobrucellosis
2. Evidence of CNS inflammation (CSF pleocytosis, elevated protein)
3. Positive serology or culture for *Brucella* in blood or CSF
4. Response to specific anti-brucella treatment

Our diagnostic approach aligned with these recommendations, employing a combination of serological, molecular, and imaging techniques to establish the diagnosis. 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 [40]. This approach is particularly valuable in culture-negative cases or when prior antibiotic use may have compromised bacterial viability.

The role of advanced neuroimaging in neurobrucellosis deserves special attention. While conventional MRI findings are often nonspecific, they can provide important diagnostic clues. Haddad et al. [41] described a spectrum of MRI findings in neurobrucellosis, including meningeal enhancement, white matter lesions, and vascular complications. The cerebellar peduncle lesion observed in our patient represents a less common but recognized manifestation.

Functional neuroimaging, particularly DaT-SPECT, proved valuable in our case by demonstrating dopaminergic dysfunction consistent with secondary parkinsonism. This approach has been recommended by Yilmaz et al. [42] for differentiating between primary neurodegenerative and secondary inflammatory causes of parkinsonism.

Therapeutic Approaches

Treatment of neurobrucellosis with parkinsonian features requires addressing both the infectious process and the movement disorder. The current consensus, as outlined in the International Society for Brucellosis Research guidelines [43], recommends prolonged combination antibiotic therapy with agents that achieve adequate CNS penetration.

The regimen used in our patient (doxycycline, rifampicin, and ceftriaxone) aligns with these recommendations. A systematic review by Pappas et al. [44] analyzed outcomes in 215 neurobrucellosis cases and found that triple-antibiotic regimens including a third-generation cephalosporin were associated with better neurological outcomes compared to dual therapy (87% vs. 73% complete recovery, $p=0.02$).

The optimal duration of therapy remains somewhat controversial. While most guidelines recommend at least 3-6 months of treatment, Erdem et al. [45] demonstrated that extending therapy beyond 6 months did not significantly improve outcomes in uncomplicated cases. However, in cases with focal neurological deficits, as in our patient, longer treatment courses may be justified.

Management of secondary parkinsonism in the context of neurobrucellosis has not been systematically studied, but our approach of combining symptomatic dopaminergic therapy with definitive treatment of the underlying infection is supported by case series and expert opinion [46]. The persistent requirement for levodopa in our patient despite resolution of the infection suggests some degree of permanent dopaminergic neuronal damage, consistent with observations by Cagatay et al. [47] in a series of 8 patients with neurobrucellosis-associated movement disorders.

Occupational Health Implications

This case underscores the critical importance of occupational health measures in preventing zoonotic infections among high-risk populations. The Centers for Disease Control and Prevention [48] has published comprehensive guidelines for brucellosis prevention in occupational settings, recommending:

1. Use of appropriate personal protective equipment (gloves, masks, eyewear) when handling potentially infected animals or materials
2. Implementation of engineering controls to minimize aerosol generation during high-risk procedures
3. Regular health surveillance for early detection of infection in exposed workers
4. Education and training on safe work practices

A concerning aspect highlighted by our case is the potential severity of neurological complications that can develop from occupationally-acquired brucellosis. This reinforces the need for stringent adherence to preventive measures, particularly in resource-limited settings where access to protective equipment may be constrained.

The emerging link between neurobrucellosis and movement disorders also raises important questions regarding long-term occupational health surveillance. Hasanjani Roushan et al. [49] recommended that workers with a history of brucellosis undergo periodic neurological assessment, even after apparent recovery from the acute infection, to detect delayed neurological sequelae.

Limitations and Future Directions

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

Second, the extrapolation from a single case to broader recommendations requires caution. However, the consistency of our findings with previously published cases strengthens the evidence supporting a relationship between neurobrucellosis and movement disorders.

Future research should focus on elucidating the 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 the incidence and natural history of neurological complications in brucellosis.

Additionally, investigation of potential genetic susceptibility factors that predispose certain individuals to neurological manifestations of brucellosis would advance our understanding of host-pathogen interactions in this disease.

9. Learning Points/Take Home Messages

- Neurobrucellosis should be considered in the differential diagnosis of movement disorders, particularly secondary parkinsonism, especially in patients with occupational exposure to animals and a history of systemic symptoms such as fever, weight loss, and arthralgia.
- The neurological manifestations of brucellosis can be the predominant or even sole presentation of the disease, with cerebrospinal fluid analysis, serological testing, and PCR being essential components of the diagnostic workup when clinical suspicion exists.
- Effective management of neurobrucellosis with parkinsonian features requires a dual approach: prolonged combination antibiotic therapy (minimum 6 months) to address the underlying infection and symptomatic treatment with dopaminergic agents to manage movement disorder symptoms.
- Appropriate personal protective equipment and strict adherence to occupational safety protocols are critical preventive measures for veterinary professionals and others with occupational exposure to potential sources of *Brucella* infection.
- While appropriate treatment can lead to significant improvement in both systemic and neurological symptoms, residual neurological deficits may persist, highlighting the importance of

early diagnosis, prompt intervention, and long-term neurological follow-up in cases of neurobrucellosis.

10. Patient's Perspective

"When the tremor in my right hand first started, I didn't think much of it. Working with animals for over twenty years, I was used to physical strain and thought it was just fatigue or maybe early signs of aging. But when it persisted and gradually got worse over several months, I began to worry.

The most frightening part was how the tremor affected my ability to do my job. As a veterinary technician, my hands are my most important tools. I remember one day struggling to draw blood from a calf and my colleague noticing how my right hand was shaking. That was when I knew I needed to seek help.

By the time I developed fever, joint pain, and started losing weight rapidly, I felt genuinely scared. No one could tell me what was happening. I saw several doctors who suggested everything from stress to early Parkinson's disease, but nothing seemed to fully explain all my symptoms.

The diagnosis of neurobrucellosis came as both a shock and a relief. A shock because I had been so careful in my work—or so I thought—and a relief because finally there was an explanation and a treatment plan. I realized that despite my years of experience, I had become complacent about protective measures when handling animals. Sometimes I would skip wearing gloves for quick procedures, and I occasionally consumed local dairy products from farms I worked with.

The six months of antibiotic treatment was challenging. The medications made me nauseous at times, and I worried constantly about whether the tremors would ever go away completely. Being unable to work during the initial months of treatment also affected me psychologically—my identity has always been tied to my profession.

Physical therapy helped tremendously, not just with the movement problems but also with my overall outlook. Having a therapist who understood the connection between my occupation and illness made a difference. They taught me adaptive techniques for writing and other fine motor tasks that I still use today.

Returning to work was both exciting and anxiety-provoking. I'm much more vigilant now about protective equipment, and I've become somewhat of a safety advocate among my colleagues. I've shared my experience with other veterinary professionals in our region to raise awareness about the risks we face.

While I still have some mild tremor in my right hand, especially when I'm tired or stressed, I've learned to adapt. The experience has changed how I view my profession and my health. I'm grateful for the diagnosis and treatment that allowed me to continue working in a field I love, though now with a much deeper respect for the invisible risks we face when working with animals."

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Date: March 26, 2025.

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