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Spondylodiscitis Caused by Salmonella enterica in an Immunocompromised Patient

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Abstract

Spondylodiscitis caused by *Salmonella enterica* is a rare manifestation of extraintestinal infection. We report the case of a 71-year-old man receiving prolonged corticosteroid therapy who was initially admitted with *S. enterica* serovar Choleraesuis bacteremia and persistent low back pain. Lumbar CT and contrast-enhanced MRI revealed a fracture of the superior endplate of L4, with associated vertebral marrow and subtle paravertebral inflammatory changes, initially interpreted as predominantly post-traumatic. Following antimicrobial therapy and clearance of blood cultures, the patient improved clinically, although low back pain persisted. Approximately two months later, he was readmitted with worsening low back pain, paresthesia, and lower limb weakness, and *S. enterica* serovar Choleraesuis was again isolated from blood cultures. MRI and fluorine-18 fluorodeoxyglucose PET/CT demonstrated L3-L4 spondylodiscitis with paravertebral extension, a posterior epidural collection, and a right psoas abscess. He was managed conservatively with approximately six weeks of ciprofloxacin, with favorable clinical, laboratory, and radiological evolution. At three-month follow-up, he was pain-free and able to walk with the aid of a cane. This case highlights the diagnostic challenges of vertebral infection in patients with *Salmonella* bacteremia, particularly when a concomitant vertebral fracture provides an alternative explanation for persistent back pain. Vertebral involvement should be considered when localized symptoms persist despite initial antimicrobial treatment and apparent clinical improvement.

Categories: Internal Medicine, Infectious Disease
Keywords: bacteremia, choleraesuis, salmonella enterica, spondylodiscitis, vertebral osteomyelitis

Introduction

Infectious spondylodiscitis usually results from hematogenous spread of microorganisms to the vertebrae and adjacent intervertebral disc space. Diagnosis may be delayed because the presentation is often insidious and dominated by persistent axial pain, while fever may be absent. MRI is the imaging modality of choice when vertebral infection is suspected [1].

Osteoarticular infection caused by *Salmonella* is rare, with osteomyelitis estimated to occur in approximately 0.8% of all *Salmonella* infections [2]. Although classically associated with sickle cell disease, it has also been described in patients with other predisposing factors, including diabetes mellitus, autoimmune disease, and immunodeficiency, as well as in adults without identifiable risk factors [2,3]. The spine is one of the possible sites of infection, with the lumbar region being most commonly involved in reported cases of *Salmonella* vertebral osteomyelitis [3].

Among non-typhoidal *Salmonella*, *Salmonella enterica* serovar Choleraesuis has a particular propensity to cause bacteremia and extraintestinal infection [4]. This swine-adapted serovar has been reported predominantly in Asia and is relatively uncommon in Europe [4]. We report the case of a patient receiving prolonged corticosteroid therapy who presented with *Salmonella* bacteremia and persistent low back pain, in whom a concomitant vertebral fracture complicated the initial recognition of vertebral infection.

This case was previously presented as a poster at the 22nd European Congress of Internal Medicine, held in Istanbul, Turkey, from March 6 to 9, 2024.

Case Presentation

A 71-year-old man with a medical history of obesity, hyperuricemia, splenomegaly, and thrombocytopenia of uncertain etiology, previously presumed to be immune thrombocytopenia, was receiving prolonged corticosteroid therapy. Prednisolone had been initiated at 20 mg/day approximately five months before the first admission, increased to 40 mg/day after eight weeks, and subsequently to 60 mg/day approximately three weeks later. The latter dose was maintained for several months. He was not receiving any other immunosuppressive agents.

Approximately two months before the current admission, he had been admitted with fever, low back pain, and pain in the right lower limb. The low back pain had been present for approximately six weeks, reportedly

beginning after physical exertion while helping his wife up from the floor at home, and had progressively worsened despite analgesia. No specific history of direct spinal trauma was documented. He reported no gastrointestinal symptoms. Laboratory tests revealed elevated inflammatory markers and severe thrombocytopenia (Table *1*). Blood cultures grew *S. enterica* serovar Choleraesuis, resistant to ampicillin and amoxicillin/clavulanic acid and susceptible to cefotaxime, ciprofloxacin, trimethoprim-sulfamethoxazole, and piperacillin/tazobactam.

Laboratory parameter	Initial admission	Readmission	Reference range
C-reactive protein (mg/dL)	20.07	33.56	<0.50
Procalcitonin (ng/mL)	9.61	3.4	0-0.5
Erythrocyte sedimentation rate (mm/h)	47	56	1-20
White blood cell count (× 10^9/L)	4.2	3	3.60-10.5
Hemoglobin (g/dL)	12.2	10.6	11.8-15.8
Platelet count (× 10^9/L)	42	28	150-450

TABLE 1: Laboratory findings at initial admission and readmission.

Comparison of laboratory results obtained during the patient's two hospital admissions. Reference ranges correspond to those used by the hospital laboratory.

Regarding epidemiological exposure, the patient reported contact with chickens and ducks, consumption of home-produced eggs, and occupational exposure to meat through his work in a butcher's shop, from which he also obtained meat for personal consumption. No stool cultures were performed, and no specific source of *Salmonella* infection could be established. Abdominal ultrasonography, performed to investigate a potential infectious source, raised suspicion of subacute appendicitis due to appendiceal wall thickening and surrounding inflammatory changes. However, subsequent abdominopelvic CT showed no evidence of appendiceal inflammation.

Non-contrast CT of the lumbar spine showed a fracture of the superior endplate of L4, with less than 50% loss of vertebral body height and no significant retropulsion of the posterior wall. No definite CT findings of spondylodiscitis were identified. Contrast-enhanced MRI, including T1-weighted, T2-weighted, STIR, and post-gadolinium fat-suppressed sequences, showed central depression involving the superior endplate of L4, with approximately 25% loss of vertebral body height, associated with T2 hyperintensity, T1 hypointensity, and contrast enhancement involving the vertebral body and left pedicle, with subtle extension into the adjacent anterior and lateral paravertebral tissues. No significant spinal canal or foraminal compromise was identified. The initial MRI report did not describe inflammatory changes involving the L3-L4 disc or the adjacent L3 vertebral endplate. Given the onset of pain following physical exertion and the presence of a vertebral fracture, these findings were initially interpreted as predominantly post-traumatic, although an underlying infectious process could not be definitively excluded.

He received ceftriaxone for two days, followed by ciprofloxacin for 16 days, initially administered intravenously at 400 mg twice daily for nine days and subsequently orally at 500 mg twice daily for seven days. Follow-up blood cultures were negative, and both clinical and laboratory improvement were observed. He was discharged.

Over the following weeks, however, the low back pain persisted and progressively worsened, with the development of paresthesia and lower limb weakness. By the time he returned to the emergency department, he was no longer able to walk independently. He remained afebrile and reported no gastrointestinal symptoms. On physical examination, he was alert, cooperative, and oriented, with pale skin and mucous membranes and bilateral lower limb edema. Cardiopulmonary examination was unremarkable. Neurological symptoms included bilateral lower limb weakness and impaired mobility, although detailed muscle strength grading, reflexes, sensory examination, and sphincter function were not documented. Laboratory tests revealed elevated inflammatory markers, leukopenia, anemia, and severe thrombocytopenia (Table *1*). New blood cultures were collected, and empirical treatment with piperacillin/tazobactam was started.

Blood cultures again grew *S. enterica* serovar Choleraesuis, with the same antimicrobial susceptibility profile as the previous isolate. After nine days of piperacillin/tazobactam, antimicrobial therapy was switched to ciprofloxacin, initially administered intravenously at 400 mg twice daily and subsequently orally at 500 mg twice daily, without overlap between the two antibiotics. Follow-up blood cultures were negative.

Lumbar spine MRI showed inflammatory changes involving the L3-L4 intervertebral space and the right posterior paravertebral tissues (Figure *1A*-*1D*), associated with the previously identified L4 fracture. Fluorine-18 fluorodeoxyglucose (18F-FDG) PET/CT demonstrated markedly increased uptake at the adjacent L3 and L4 vertebral endplates (SUVmax 14.3), with extension into the paravertebral soft tissues, particularly on the right, and involvement of the right psoas muscle (SUVmax 16.4), findings consistent with spondylodiscitis (Figure *2A*-*2D*). Small hypodense areas with peripheral uptake were also identified within the psoas muscle, raising suspicion of abscess formation. A focal area of increased uptake was also identified in the transverse colon (SUVmax 13.5).

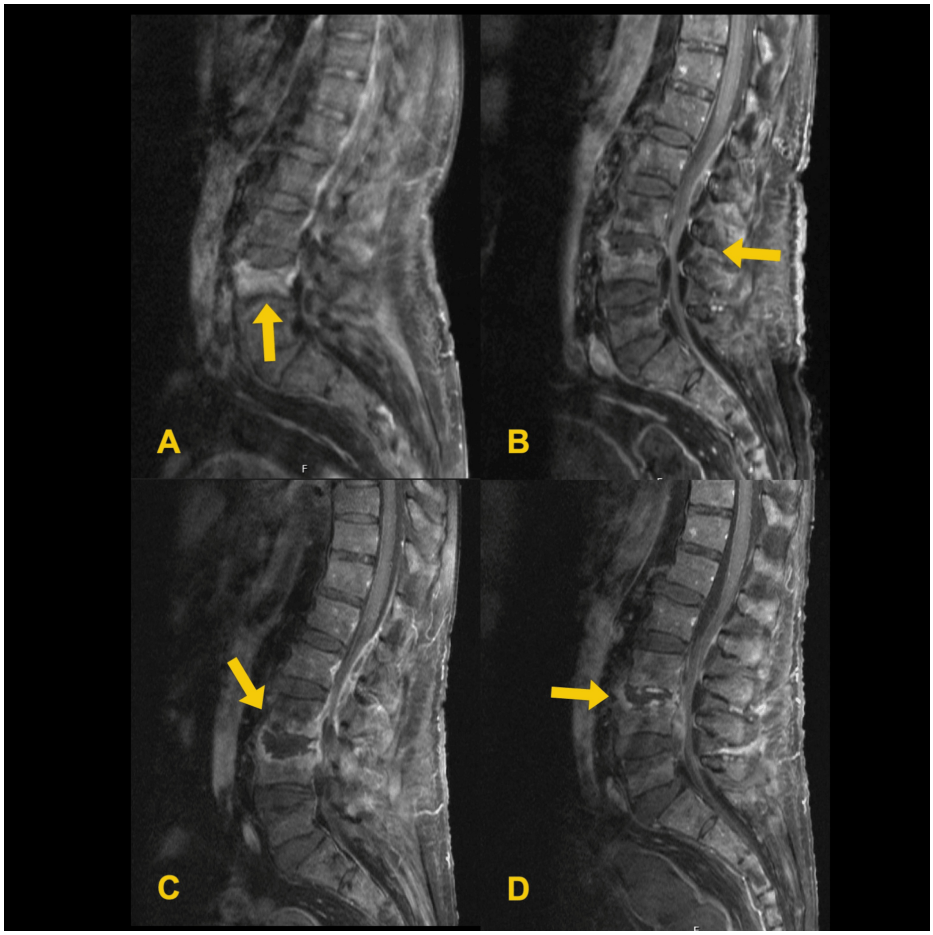


FIGURE 1: Serial lumbar spine MRI demonstrating the evolution of L3-L4 spondylodiscitis.

Sagittal magnetic resonance images obtained at different stages of the disease. (A) Initial contrast-enhanced MRI demonstrating a compression fracture of the superior endplate of L4, initially interpreted as predominantly post-traumatic. (B) Subsequent MRI showing more pronounced inflammatory changes involving the posterior elements of L3-L4 and adjacent paravertebral tissues. (C) Repeat MRI demonstrating progressive inflammatory and destructive changes involving the L3-L4 intervertebral disc and adjacent vertebral endplates, consistent with spondylodiscitis. (D) Follow-up MRI during antimicrobial treatment showing persistent discovertebral abnormalities despite clinical improvement. Yellow arrows highlight the principal abnormalities. The L3-L4 level is identified by the intervertebral disc immediately superior to the fractured L4 vertebral body.

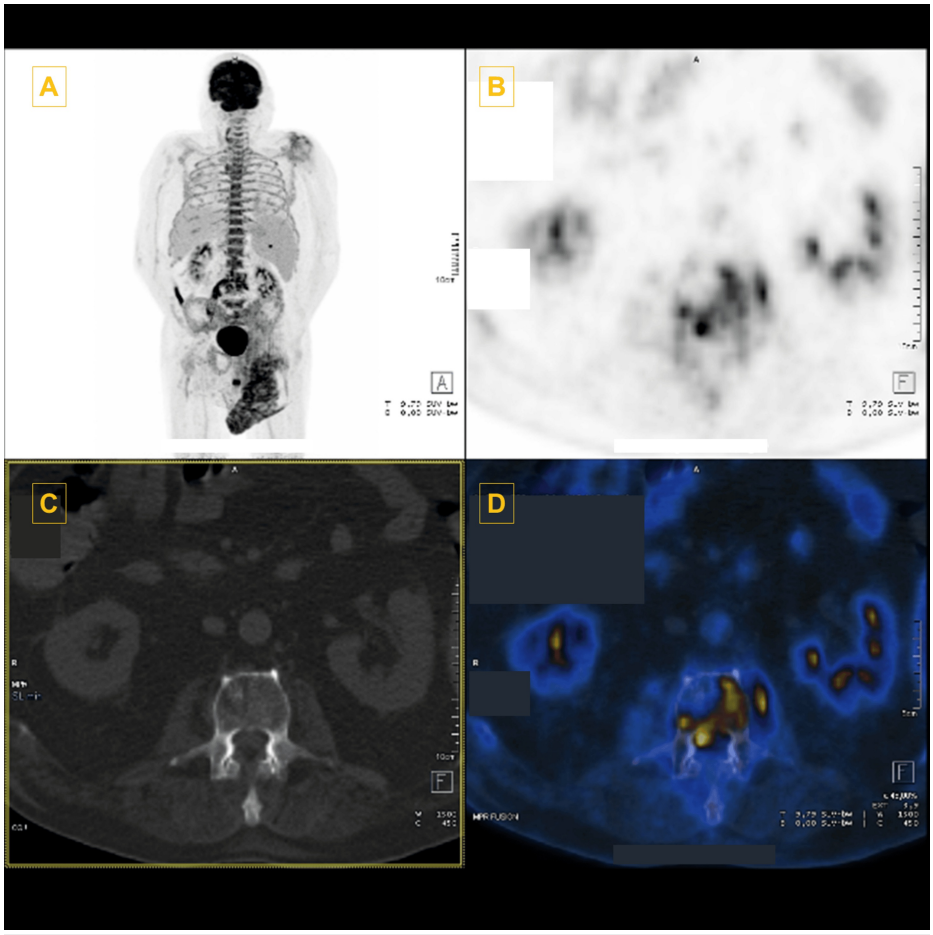


FIGURE 2: 18F-FDG PET/CT findings in L3-L4 spondylodiscitis.

18F-FDG PET/CT images demonstrating the distribution of abnormal radiotracer uptake. (A) Whole-body maximum intensity projection showing increased metabolic activity in the lumbar region. (B) Coronal PET image demonstrating increased uptake in the lumbar paravertebral region. (C) Axial CT image at the level of the lumbar spine. (D) Corresponding axial fused PET/CT image demonstrating increased metabolic activity involving the vertebral and adjacent paravertebral tissues, particularly on the right, consistent with infectious involvement. The PET/CT examination also identified increased uptake extending into the right psoas muscle and a separate focal area of uptake in the transverse colon, as described in the Case Presentation.

18F-FDG, fluorine-18 fluorodeoxyglucose

Given the clinical and imaging evolution, a repeat MRI was performed, showing more pronounced inflammatory changes involving the L3-L4 disc and perivertebral tissues, together with an abscess in the right psoas muscle and a small posterior epidural collection (Figure *1*). Transthoracic echocardiography showed no vegetations or other findings suggestive of endocarditis. No findings suggestive of an endovascular infectious focus were reported on 18F-FDG PET/CT, although the examination was not specifically directed at excluding aortic infection.

The case was evaluated repeatedly by Neurosurgery. Although the patient reported lower limb weakness and impaired mobility, no focal neurological deficits were reported by the neurosurgical team during the assessment. Despite the presence of paravertebral and epidural collections, conservative management was recommended, with continued antimicrobial therapy, close clinical monitoring, and serial MRI examinations. A vertebral biopsy was not performed. Given the repeated isolation of *S. enterica* serovar Choleraesuis from blood cultures and the compatible clinical and imaging findings, antimicrobial treatment was initiated without awaiting vertebral tissue sampling. As the patient subsequently showed a favorable clinical and radiological response, a vertebral biopsy was not pursued. Given the prolonged exposure to high-dose corticosteroids, prednisolone was gradually tapered rather than abruptly discontinued.

The focal uptake identified in the transverse colon prompted colonoscopy, which revealed a sessile polypoid lesion that was removed by piecemeal mucosal resection. Histopathological examination showed a tubulovillous adenoma with low- and high-grade dysplasia, with no evidence of invasion of the muscularis mucosae in the sections examined. No causal relationship between this finding and the *Salmonella* infection was established.

Follow-up MRI showed a marked reduction in the posterior epidural collection and right psoas collections, together with improvement in the disc and paravertebral inflammatory changes (Figure *1*). No significant neural compression or vertebral instability requiring surgical intervention was identified. He completed approximately six weeks of ciprofloxacin therapy, calculated from the first day of ciprofloxacin administration, with favorable clinical, laboratory, and radiological evolution, and was discharged to a rehabilitation unit.

At clinical follow-up three months after admission, he was pain-free and able to walk independently with the aid of a cane.

Discussion

S. enterica serovar Choleraesuis differs from other non-typhoidal serovars in its particular propensity to cause bacteremia and invasive extraintestinal disease [4]. This feature is particularly relevant in the present case. In a series of 129 adults with non-typhoidal *Salmonella* bacteremia, 51 (39.5%) developed extraintestinal focal infections, with vertebral osteomyelitis among the most frequent sites [5]. Age was independently associated with the development of focal infection in that population [5]. Other studies have also reported a higher frequency of osteomyelitis and other invasive complications of non-typhoidal *Salmonella* bacteremia in older patients [6].

In this patient, advanced age and prolonged corticosteroid therapy provided a clinical context favoring invasive disease. The association between older age or immunocompromised status and invasive non-typhoidal *Salmonella* infection is well established, although vertebral involvement cannot be directly attributed to corticosteroid therapy [4,6].

The source of infection remained uncertain. The patient reported contact with domestic poultry, consumption of home-produced eggs, and occupational exposure to meat through his work in a butcher's shop. Although these exposures may represent potential routes of *Salmonella* acquisition, none could be established as the source of infection. In particular, *S. enterica* serovar Choleraesuis is predominantly associated with swine, and no specific exposure could be directly linked to the infection. No stool cultures were performed, limiting the investigation of a possible gastrointestinal source. Abdominal ultrasonography initially raised suspicion of appendiceal inflammation, but this finding was not confirmed on subsequent CT. Therefore, a definitive source of infection could not be established.

One of the most relevant aspects of this case was the difficulty in recognizing vertebral involvement during the first episode of bacteremia. The patient already had persistent low back pain, but an L4 fracture following physical exertion provided a plausible explanation for the pain and some of the abnormalities seen on MRI. The initial examination demonstrated vertebral marrow signal abnormalities and enhancement involving the superior endplate of L4, with subtle extension into the adjacent paravertebral tissues. Although these findings were initially interpreted as predominantly post-traumatic, they were also compatible with an early infectious process, particularly in the presence of *Salmonella* bacteremia and elevated inflammatory markers. The initial radiology report did not describe inflammatory changes involving the L3-L4 disc or the adjacent L3 vertebral endplate, which may have contributed to the diagnostic uncertainty. Vertebral osteomyelitis may present with nonspecific symptoms and can initially be mistaken for mechanical or degenerative spinal disease [1].

In retrospect, the persistence of low back pain after the first admission, followed by recurrent bacteremia with the same serovar and progression of the L3-L4 abnormalities, with subsequent development of psoas and epidural collections, makes it plausible that vertebral infection was already present during the first episode. Nevertheless, this cannot be demonstrated definitively, since no vertebral sample was obtained for microbiological analysis. Furthermore, although the same serovar with an identical antimicrobial susceptibility profile was isolated during both admissions, molecular typing was not performed. Consequently, relapse from an unresolved infectious focus cannot be definitively distinguished from reinfection associated with ongoing epidemiological exposure. If vertebral infection was already established during the first admission, the initial 18-day course of antimicrobial therapy would have been substantially shorter than the six weeks recommended for most cases of bacterial vertebral osteomyelitis [1].

During the second admission, the repeated isolation of *S. enterica* serovar Choleraesuis from blood cultures, together with the progression of the imaging findings, strongly supported the diagnosis of spondylodiscitis caused by this organism. Although no vertebral biopsy was obtained, the microbiological and imaging findings, considered together, provided strong support for this etiological diagnosis. Antimicrobial treatment was initiated without delaying therapy for vertebral tissue sampling, and the subsequent favorable response supported continuation of the established treatment strategy. Nevertheless, the absence of vertebral tissue sampling precludes direct microbiological confirmation at the spinal site. MRI remains the imaging modality of choice when vertebral osteomyelitis is suspected and allows assessment of disease extension into the paravertebral tissues and epidural space [1].

The possibility of endovascular infection also deserves consideration, particularly given the invasive nature

of *S. enterica* serovar Choleraesuis and the recurrence of bacteremia. Transthoracic echocardiography did not reveal findings suggestive of endocarditis, and no suspicious endovascular focus was described in the 18F-FDG PET/CT report. However, these findings do not definitively exclude endovascular involvement, particularly in the absence of dedicated vascular imaging.

Ciprofloxacin was used according to the susceptibility profile of the isolate. Fluoroquinolones have high oral bioavailability and are a particularly useful option for vertebral osteomyelitis caused by susceptible Gram-negative bacilli [1]. However, the literature specifically addressing *Salmonella* osteomyelitis shows considerable heterogeneity in the antimicrobials used and treatment duration, reflecting the rarity of the condition and the absence of trials specifically addressing this pathogen [2]. The approximately six-week course of ciprofloxacin used in this case, calculated from the first day of its administration during the second admission, is consistent with the recommended duration for most cases of bacterial vertebral osteomyelitis [1].

Despite the presence of epidural and psoas collections, a conservative approach was possible. In vertebral osteomyelitis, surgical intervention is mainly considered in patients with progressive neurological deficits, vertebral deformity or instability, symptoms of spinal cord compression, or progression or recurrence of infection despite appropriate antimicrobial therapy [1]. In this case, repeated neurosurgical evaluations supported continued medical treatment with close clinical monitoring and serial MRI examinations. Although the patient reported lower limb weakness and impaired mobility, detailed neurological examination findings were not available in the clinical records. Subsequent imaging demonstrated a favorable response, without significant neural compression or vertebral instability requiring surgical intervention.

Conclusions

This case illustrates the diagnostic challenges of vertebral osteomyelitis in patients with non-typhoidal *Salmonella* bacteremia, particularly when a concomitant vertebral fracture provides an alternative explanation for persistent back pain. The initial clinical improvement following antimicrobial therapy did not exclude an underlying vertebral infection, which became apparent after symptom progression and recurrent bacteremia.

Persistent or worsening back pain in patients with *Salmonella* bacteremia should prompt consideration of vertebral involvement, particularly in older or immunocompromised individuals. Vertebral endplate enhancement and adjacent paravertebral inflammatory changes on MRI should not be attributed solely to trauma in this clinical context. Early recognition and appropriate antimicrobial treatment are essential to prevent disease progression and potentially serious complications.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Tiago Jorge Costa, João Ricardo Machado, Daniela Maurício

Acquisition, analysis, or interpretation of data: Tiago Jorge Costa, João Ricardo Machado, Daniela Maurício, Joana Cascais Costa, João Costelha

Drafting of the manuscript: Tiago Jorge Costa, Daniela Maurício

Critical review of the manuscript for important intellectual content: Tiago Jorge Costa, João Ricardo Machado, Joana Cascais Costa, João Costelha

Supervision: Joana Cascais Costa, João Costelha

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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