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Concomitant *Listeria monocytogenes* and *Streptococcus equinus* brain abscess in an immunocompetent individual: a case report

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Abstract

Background *Listeria monocytogenes* brain abscess is a rare phenomenon that is common in immunocompromised patients. *Streptococcus equinus* brain abscess has never been reported in the literature to our knowledge. In this case report, we describe a case of brain abscess secondary to *Listeria monocytogenes* and *Streptococcus equinus* in an immunocompetent patient with transient low CD4 count.

Case presentation A 27-year-old white, male patient, previously healthy, nonalcoholic, and occasional smoker, presented to the emergency department for confusion and headache. The patient was found to have a left parietal abscess, which was drained and the fluid was sent for culture. Culture grew *Listeria monocytogenes* and *Streptococcus equinus*. The patient was treated with intravenous ampicillin followed by oral amoxicillin for a total of 6 weeks. The CD4 count was low initially. However, after the resolution of the infection, the CD4 count came back within normal range. Another brain magnetic resonance imaging was done that showed a significantly decreased hyperintensity within the left parietal subcortical white matter at the site of surgery with significantly decreased enhancement and almost total resolution of the previous abscess.

Conclusion Transient low CD4 count is a rare phenomenon that exposes patients to unusual and atypical infections. Since low CD4 count is transient, patients treated promptly recover from their illness. Our patient developed a *Listeria monocytogenes* and *Streptococcus equinus* brain abscess, which is considered rare and has not been previously described in the literature to our knowledge.

Keywords Brain abscess, *Listeria monocytogenes*, *Streptococcus equinus*, Immunocompetent patient

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Introduction

Idiopathic $CD4^+$ T-cell lymphocytopenia (ICL) is a rare immune system disorder characterized by diverse clinical manifestations. It was initially defined by the Centers for Disease Control and Prevention in 1992 as a CD4 count below 300 cells/mm^3 in the absence of human immunodeficiency virus infection [1]. The exact cause of ICL remains unclear [2]. In some instances, low $CD4^+$ T lymphocyte counts may result from transient responses to infections or other conditions, or even represent a normal clinical status in asymptomatic patients [3, 4]. Its progression differs from acquired immunodeficiency syndrome, although individuals with ICL may experience opportunistic infections [1]. Noteworthy opportunistic infections reported in the literature include diseases associated with human papillomavirus, molluscum contagiosum, cryptococcosis, and nontuberculous mycobacterial infections [5].

In our case, we presented the hospital course of an immunocompetent patient with a transient decline in CD4 count who developed a brain abscess due to *Listeria monocytogenes* and *Streptococcus equinus*. The patient underwent surgical drainage and received intravenous ampicillin, followed by oral amoxicillin for a total of 6 weeks, resulting in complete resolution of the opportunistic infection. Subsequently, the patient's CD4 count returned to normal levels.

Case presentation

A 27-year-old white, male patient, previously healthy, nonalcoholic, and occasional smoker, presented to the emergency department for confusion. Three days prior to presentation, the patient started complaining of episodic headaches, fluctuating in intensity, partially relieved by over-the-counter pain medications (paracetamol 1 g per os every 6 hours and ibuprofen 400 mg per os every 8 hours as needed for 3 days). His headache gradually worsened and was followed by a sudden onset of confusion on the day of admission.

There was no reported fever, chills, loss of consciousness, nausea, vomiting, abnormal movements, urinary or fecal incontinence, or history of head trauma or fall, no recent dental procedure was performed except for an uncomplicated tooth extraction 5 years prior to presentation, and there was no reported recent upper respiratory tract infection. He denied any drug abuse or any high-risk sexual behavior. Travel history was pertinent for a trip to Italy 4 months prior to presentation. He works as a business man and lives with his wife in an urban area where they are not regularly exposed to fumes, chemicals, or other pollutants. Family history is negative for any chronic diseases.

Upon arrival to the emergency department, his blood pressure was 131/68 mmHg, heart rate was 98 beats per minute, and he was afebrile with a temperature of 36.5 °C. On neurological examination, the patient was conscious, not oriented to people, time, or place. Cranial nerve examination was unremarkable. His pupils were equal and reactive. No nystagmus was identified. No nuchal rigidity, focal neurologic deficits, or Babinski sign were noted. His speech was normal. Motor power in all four limbs, sensory examination (light touch, pain, temperature, vibration, and proprioception), all reflexes, and gait were intact. No abnormal movements were observed. His physical examination was otherwise unremarkable, showing no signs of sinusitis, otitis, or mastoiditis. Chest rise was symmetrical, and lungs were clear to auscultation without any added sounds. Heart sounds were normal, and no murmurs were heard. His abdomen was soft to palpation and nontender, normal bowel sounds were heard, and he had no hepatosplenomegaly. Pulses were symmetrical in all four limbs, no lower limb edema was witnessed, and no lymphadenopathy was palpable.

Laboratory studies revealed a white blood cell (WBC) count of 11,230/μL (77% neutrophils), a hemoglobin of 13.9 g/dL, and a platelet count of 240,000/mm^3. Serum sodium was 136 mmol/L, potassium 4 mmol/L, chloride 101 mmol/L, bicarbonate 24 mmol/L, calcium 9.2 mg/dL, phosphorus 4.3 mg/dL, and magnesium 2 mg/dL. Creatinine was 0.64 mg/dL with a glomerular filtration rate (GFR) of 134 mL/minute/1.73 m^2, and liver function tests were within normal range (AST 13 units per liter, SGPT 13 units per liter, GGT 25 units per liter, alkaline phosphatase 69 units per liter, and total bilirubin 0.92 mg/dL). C-reactive protein (CRP) was slightly elevated, reaching 21 mg/dL. The reference values for the lab results are summarized in Table 1.

Nonenhanced computed tomography (CT) scan of the brain showed edema in the left parieto-occipital lobes surrounding a 3-cm isodense lesion in the parietal lobe suspicious of a tumor versus an infectious process, without midline shift. A total body scan ruled out any malignant process or distant infection.

Magnetic resonance imaging (MRI) of the brain with gadolinium was performed next and revealed a left parietal ring enhancing space-occupying lesion in the left parietal area measuring $3.9 \times 3.7 \times 2.4$ cm, with irregular lobulated wall, showing significant diffusion restriction (Fig. 1a–c).

The patient underwent urgent craniotomy to discover a cyst containing yellowish secretions (after aspiration) compatible with a brain abscess. The latter lesion was grossly totally resected and sent to the microbiology laboratory immediately in a sterile container. The patient was empirically started on ceftriaxone 2 g intravenously

Table 1 Reference values for the lab results

White blood cells (WBC)	$3.5–10.5 \times 10^9$/L
Hemoglobin (Hb)	13.5–17.5 g/dL
Platelets	150,000–450,000/mm^3
Sodium	135–145 mmol/L
Chloride	3.5–5.1 mmol/L
Carbon dioxide (CO_2)	22–29 mmol/L
Calcium	8.5–10.5 mg/dL
Phosphorus	2.5–4.8 mg/dL
Magnesium	1.6–2.6 mg/dL
Creatinine	0.67–1.2 mg/dL
Aspartate aminotransferase (AST)	0–40 U/L
Alanine aminotransferase (ALT)	0–41 U/L
Gamma-glutamyl transferase (GGT)	10–71 U/L
Alkaline phosphatase	40–130 U/L
Total bilirubin	0.1–1 mg/dL
C-reactive protein (CRP)	0–5 mg/dL
Lymphocyte count	1000–4500/mm^3
T helper CD4	29–60% of lymphocyte count/mm^3
T cytotoxic CD8	19–48% of lymphocyte count/mm^3
Ratio CD4/CD8	0.6–2.8

twice daily, vancomycin 2 g intravenously once as a loading dose followed by 1.5 g intravenously twice daily, and trimethoprim–sulfamethoxazole 800/160 mg per os every 8 hours. Dexamethasone 4 mg intravenously every 8 hours was also initiated and continued for 5 days. Levetiracetam 500 mg per os twice daily was introduced for seizure prophylaxis and continued throughout the hospital stay (2 weeks). Low-molecular-weight heparin was added as well for deep venous thrombosis prophylaxis at a dose of 40 mg subcutaneously daily and continued throughout the hospital stay. The sample was cultivated on both aerobic and anaerobic media (Columbia agar, MacConkey agar, chocolate agar, Schädler medium, and thioglycollate medium) along with antibiotic sensitivity testing. Gram staining of the abscess pleomorphic catalase-negative Gram-positive bacilli and coccobacilli was performed. The API Coryne system was also used for identification of bacteria. Fungal and mycobacterial cultures were not performed. Three days later, intraoperative culture grew *Listeria monocytogenes* (on Columbia agar, MacConkey agar, chocolate agar, Schädler medium. and thioglycollate medium) sensitive to ampicillin (minimal inhibitory concentration 0.75 mg/L, sensitive cutoff 1 mg/L) and *Streptococcus equinus* (that grew on Columbia agar) sensitive to penicillin G (minimal inhibitory concentration 0.016 mg/L, sensitive cutoff 0.25–2 mg/L). One set of peripheral blood cultures for aerobic and anaerobic bacteria showed no evidence of bacteremia. Therefore, antibiotics were switched to ampicillin 2 g intravenously every 3 hours for a total of 14 days followed by oral amoxicillin 1 g every 6 hours for four additional weeks to complete 6 weeks of treatment. Oral levetiracetam was also continued at a dose of 500 mg twice daily for the following 6 months.

Meanwhile, further investigations were pursued to establish a comprehensive explanation of the attained results. Human immunodeficiency virus (HIV)-1 and HIV-2 tests were negative. Total serum immunoglobulin levels were normal with an IgG level of 756 mg/dL, IgM level of 62.3 mg/dL, and Ig level of 279 mg/dL. Finally, a T-cell profile revealed a low total lymphocyte count of 620/mm^3, a CD4 T-cell deficiency with a total CD4 count of 265, and a percentage of 21% with a CD4/CD8 ratio of 0.66. Strikingly, our patient had no history of previous infections. As a result, it was postulated that our patient could potentially suffer from idiopathic CD4 depletion, but repeat testing was necessary to exclude a transitory deficiency.

Six weeks later, a follow brain MRI showed significantly decreased previously noted edematous changes within the left parieto-occipital area surrounding the significantly smaller postsurgical cavity with significantly decreased enhancement and size of 1.8×1.2 cm with the more inferior ring-enhancing lesion/microabscess of 5 mm versus previously 12 mm. Surrounding edematous changes were also decreased (Fig. 2a–c).

Six weeks after completion of antimicrobial therapy, another brain MRI was done that showed a significantly decreased hyperintensity within the left parietal subcortical white matter at the site of surgery with significantly decreased enhancement and almost total resolution of the previously noted inferior micronodular enhancement with only residual hazy enhancement superiorly extending over around 7×5 mm (Fig. 3).

The patient was seen again at 6 weeks after surgery as well as 3 months and 6 months later, in which his physical examination and neurological examination were completely normal and he did not suffer from any sequelae.

In the end, subsequent reevaluation of the T-cell profile exhibited an increase in total lymphocyte count to 2244/mm^3 with a $CD4^+$ cell count of 745/mm^3 and a percentage of 33.2% with a CD4/CD8 ratio of 0.96, validating the diagnosis of transient CD4 T-cell deficiency.

Discussion

Brain abscess is a critical infection often identified in individuals with predisposing high-risk factors, confirmed through imaging. In our case, the patient was a previously healthy young individual who presented with headache and confusion. The diagnosis revealed idiopathic CD4 lymphopenia complicated

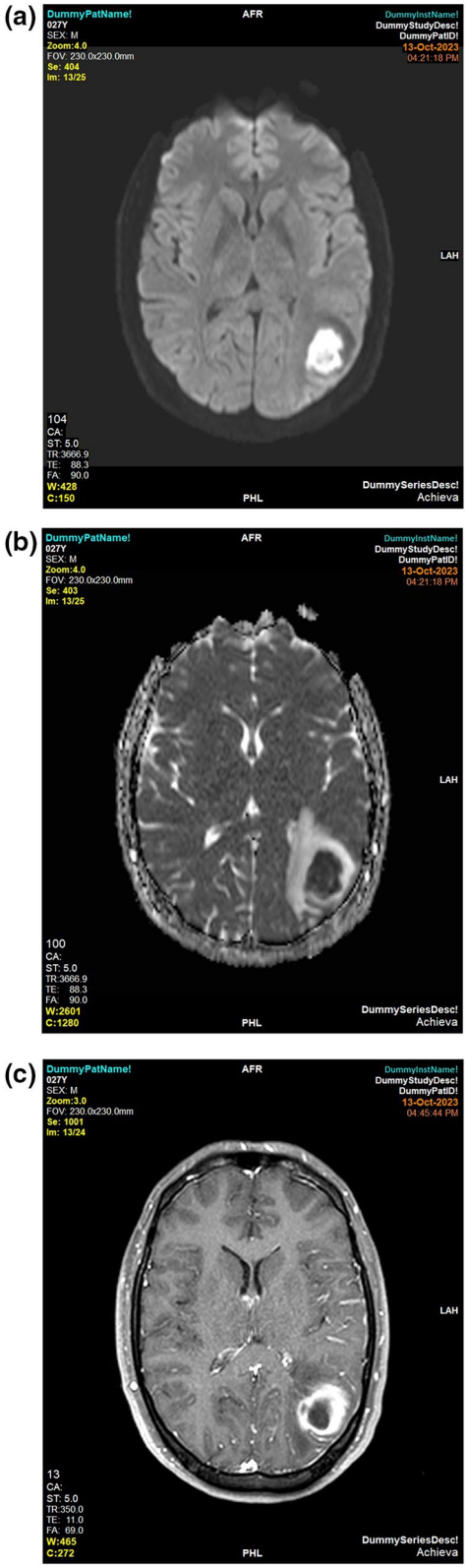

Fig. 1 **a** Diffusion weight image, with left parietal space-occupying lesion showing significant diffusion restriction surrounded by perilesional edema. **b** Apparent diffusion coefficient (ADC) mapping, with left parietal space-occupying lesion showing significant diffusion restriction surrounded by perilesional edema. **c** Post-intravenous contrast T1 Fat Saturation (FS) axial acquisition, with the same lesion showing thick ring enhancement with a hypointense center

by a polymicrobial brain abscess, which cultured *Listeria monocytogenes* and *Streptococcus equinus*. Polymicrobial brain abscesses are usually associated with oral pathogens [6]; however, our patient had no dental issues, making this case unique.

Idiopathic CD4 lymphocytopenia (ICL) was initially characterized in 1992 by the US Centers for Disease Control and Prevention (CDC) as the consistent presence of a $CD4^+$ T-lymphocyte count below 300 cells per cubic millimeter or less than 20% of total T cells. This occurs in the absence of evidence of human immunodeficiency virus (HIV) infection and without any condition causing depressed CD4 counts[11]. Investigations into cases of idiopathic depletion of $CD4^+$ T lymphocytes reveal their rarity, diverse nature, and the possibility that diminished counts of $CD4^+$ T lymphocytes could be temporary reactions to viral infections, medications, malignancies, or autoimmune diseases or sometimes normal findings in asymptomatic individuals [3, 4]. ICL is typically identified after an opportunistic infection in individuals who do not have established immunosuppression [4]. The most common opportunistic infections associated with ICL include diseases related to human papillomavirus, molluscum contagiosum, cryptococcosis, and nontuberculous mycobacterial diseases [5].

Listeria monocytogenes is a beta-hemolytic Gram-positive rod that can grow facultatively intracellularly on a specialized agar called Mueller–Hinton agar [7]. The bacterium possesses virulence factors such as internalins for host cell attachment, listeriolysin O to evade from host cell vacuoles, actin polymerization for intracellular mobility, and the capacity to replicate at refrigerated temperatures [7]. Transmission typically occurs through the fecal–oral route, often linked to the consumption of foods such as cold deli meats and unpasteurized milk products [7]. While pregnant women, neonates, the elderly, and immunocompromised individuals are more susceptible, listeriosis can also affect healthy individuals, presenting as febrile gastroenteritis [7]. On the other hand, invasive listeriosis can lead to severe clinical manifestations, including

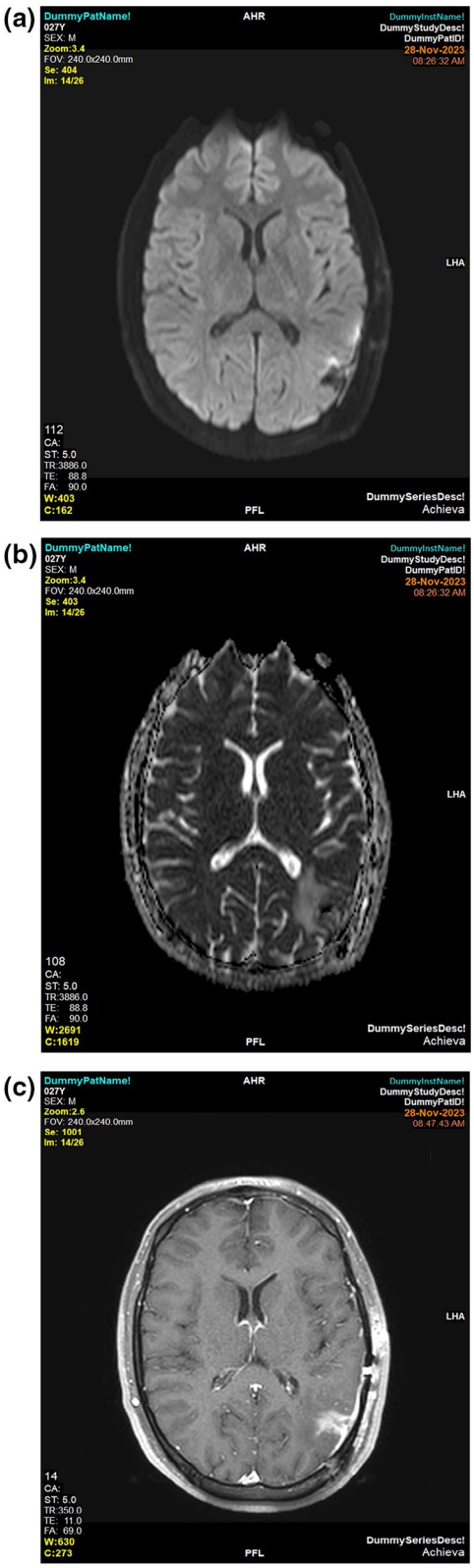

Fig. 2 **a** Diffusion weight image—no residual significant diffusion restriction noted at the site of the previously noted lesion (postsurgical changes noted with decreased edematous changes). **b** Apparent diffusion coefficient (ADC) mapping—no residual significant diffusion restriction noted at the site of the previously noted lesion (postsurgical changes noted with decreased edematous changes). **c** Post-intravenous contrast T1 Fat Saturation (FS) axial acquisition—postsurgical changes noted with significantly decreased previously noted ring-enhancing space-occupying lesion and only visible ill-defined pseudonodular enhancement at the surgical site with postsurgical meningeal enhancement and mild perilesional edema

abortion, sepsis, and meningoencephalitis [7]. Atypical clinical forms, accounting for 5–10% of cases, include endocarditis, myocarditis, arteritis, pneumonia, pleuritis, hepatitis, cholecystitis, peritonitis, and localized abscesses [8]. Brain abscesses, constituting around 10% of central nervous system infections caused by *Listeria* spp., are a notable complication [8].

Several case reports describe *Listeria*-induced brain abscesses in various patient demographics. Frade *et al.*. [9] reported multiple brain abscesses in an immunocompetent elderly individual, in contrast to our patient who was young. Manfredi *et al.*. [10] detailed a case of

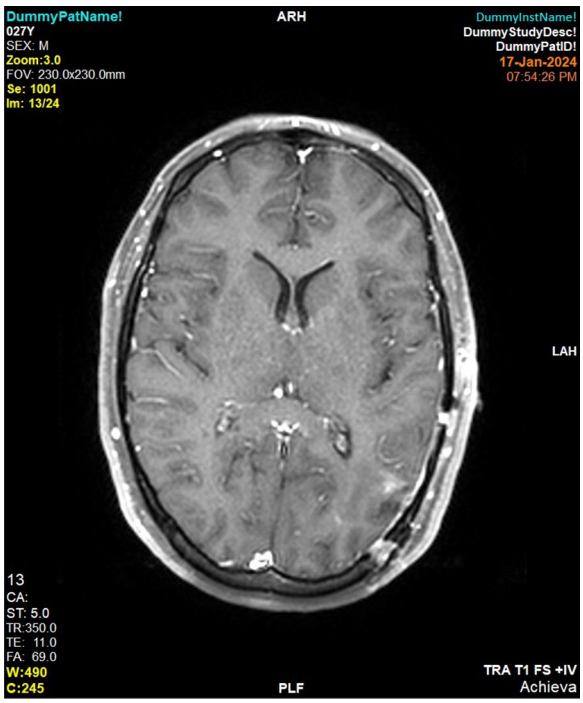

Fig. 3 Post-intravenous contrast T1 Fat Saturation (FS) axial acquisition—almost total resolution of previously noted enhancement with residual millimetric focus of enhancement, decreased enhancement at the surgical site, and significantly decreased edematous changes

Table 2 Summary of the case reports

Authors	Year of the study	Title of the study
Frade *et al.*	2020	*Multiple Listeria abscesses in an immunocompetent patient*
Manfredi *et al.*	2006	*Listeria monocytogenes meningitis and multiple brain abscesses in an immunocompetent host. Favorable response to combination linezolid-meropenem treatment*
Tiri *et al.*	2018	*Listeria monocytogenes brain abscess: controversial issues for the treatment—two cases and literature review*
Mukherjee *et al.*	2011	*Unusual presentation of brain abscess with uncommon organism in an immunocompetent person*
Leibovitch *et al.*	1991	*Multiple brain abscesses caused by Streptococcus bovis*
Sánchez, J.C., *et al.*	2023	*Streptococcus bovis infection of the central nervous system in adults: report of 4 cases and literature review*

a 50-year-old patient without any past medical history. Tiri *et al.*. [11] described a case of a 69-year-old patient previously healthy who presented with fever and rapidly deteriorating neurological function and was found to have *Listeria* brain abscess. Mukherjee *et al.*. [12] reported a case of multiple brain abscesses secondary to *Listeria* in a 55-year-old previously healthy patient who presented with progressive weakness and difficulty with micturition. A summary of those case reports can be found in Table 2.

On the other hand, *Streptococcus equinus* is non-enterococcal group D streptococcus, non-beta hemolytic Gram-positive cocci in chains that grow on Columbia blood agar [13].

Our understanding of the virulence and pathogenicity of *Streptococcus bovis*/*Streptococcus equinus* (SBSEC) is restricted to a small number of adhesion molecules and pro-inflammatory factors [14]. The *Streptococcus bovis* group, now more commonly referred to as the *Streptococcus gallolyticus* group, includes several species, one of which is *Streptococcus equinus* [15]. *S. equinus* typically colonizes the human colon [13] but rarely causes bacteremia with secondary endocarditis [16] and peritonitis [17].

Conclusion

To our knowledge, no cases of *Streptococcus equinus* brain abscess have been reported. *Streptococcus bovis*, a pathogen similar to *Streptococcus equinus*, has been reported to cause brain abscesses [18, 19]. Thus, *Streptococcus equinus* brain abscess was never reported in the literature. *Listeria* brain abscess in an immunocompetent adult as our patient is unusual. In addition, the presence of both *Listeria* and *Streptococcus equinus* was also never reported, which makes our case unique.

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Author contributions
MA and MHA wrote the case report. SH, RM, and EF reviewed the paper for intellectual content. PY, JS, and RH supervised the work. All authors critically revised the manuscript and read and approved its final version.

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Availability of data and materials
All data pertaining to this patient are included in this report.

Declarations

Ethics approval and consent to participate
An approval from an ethics committee was not needed for this case report since it involved one patient.

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

Competing interests
There authors have no conflict of interest to disclose.

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