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Neurolisteriosis with a stroke-like presentation in a male adult with polymyalgia rheumatica: a case report and literature review

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Abstract

Listeria monocytogenes is a gram-positive bacillus that can cause a wide spectrum of clinical manifestations, ranging from paucisymptomatic to life-threatening disease. Central nervous system involvement is called neurolisteriosis and is a rare and severe entity that includes meningitis, meningoencephalitis, rhombencefalitis, and brain abscess, and it may start with a stroke-like presentation. People with rheumatic diseases taking immunosuppressive medications are at increased risk, and they also can have atypical presentations, misleading the diagnosis of infection. Magnetic resonance imaging can guide the diagnosis; however, a high clinical suspicion and a microbiological confirmation are needed. Here we present a case of Listeria monocytogenes brain abscess mimicking an ischemic stroke that occurred in a male patient with a previous diagnosis of polymyalgia rheumatica under immunosuppressive treatment. A literature revision was also performed.

Key words: *Listeria monocytogenes*, neurolisteriosis, methotrexate, glucocorticoids, polymialgia reumatica, case report.

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Introduction

*Listeria monocytogenes i*s a short gram-positive, facultative anaerobic, non-spore-forming, facultative intracellular bacillus belonging to the phylum Firmicutes, class Bacilli, and order Bacillales, recognized as a human foodborne pathogen in the 1980s.[1-5] *L. monocytogenes* infection, called listeriosis, is mainly a food-borne disease contracted through the ingestion of contaminated products such as processed meat, dairy products, pre-packed sandwiches, cold-smoked fish, prepared vegetables, salads, fruits and frozen foods like ice cream among others. Transmission follows the fecal-oral route; many cases are sporadic, but food-borne outbreaks are observed.[3] *L. monocytogenes* remains one of the main cuases of food-borne diseases with the highest mortality rate,[3,6,7] with an estimated incidence ranging from 0.1 to 11.3 cases per million population per year, depending on the geographic region.[3] *L. monocytogenes* has many virulence factors: it can bind primary to gastrointestinal epithelial cells *via* cadherin entry, spreading through the cytosol and cell-to-cell (thus escaping host antibody response), producing biofilms and disseminate hematogenously.[3,7] Moreover, it can be spread by the macrophages throughout the reticulo-endothelial system, and it can penetrate the blood-brain barrier as well as the placenta-fetus barrier.[3,7-12]

Listeriosis spectrum ranges from subclinical, or mild self-limiting, febrile gastroenteritis (in particular in immunocompetent hosts) to invasive, potentially deadly, disease.[3,7,13] Risk factors for invasive disease are pregnancy, extreme ages (*e.g*. neonates and elderly), cancer or other causes of immunosuppression. i) Invasive disease; ii) septicemic listeriosis; iii) central nervous system (CNS) infection called neurolisteriosis (NL). Less common infection sites include the peritoneal cavity, musculoskeletal system, eyes, cardiovascular system, urinary and biliary tract, and skin, among others.[3]

Polymyalgia rheumatica (PMR) is a systemic, inflammatory, multifactorial, rheumatic disorder that affects people older than 50 with a female:male ratio of 2-3:1 and is more frequent in people of northern descent.[14] It is characterized by musculoskeletal inflammatory pain in the pelvic and scapular girdle and neck variably associated with fatigue, arthralgia, lack of appetite, weight loss, non-erosive synovitis, and fever. In up to 30% of cases it can be associated with giant cell arteritis (GCA), and PMR occurrs in up to 50% of patients with GCA.[14,15] According to the American College of Rheumatology/European League Against Rheumatism recommendations, treatment of PMR is primarily based on glucorticoids (GCs) with a gradual tapering according to clinical response and, in patients with relapsing/refractory PMR or with intolerance to GCs due to comorbidities or with poor prognostic factors, conventional synthetic disease-modifying anti-rheumatic drugs (DMARDs) such as methotrexate (MTX) or biologic (b) DMARDs, such as anti-interleukin (IL)-6 compounds, with the aim of a steroid-sparing effect.[16-18]

PMR treatments are associated with increased infection risk, and also GC usage is associated with a relative and absolute

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increased risk of all-cause bacterial, viral, fungal, and parasitic infections in a dose- and time-dependent manner with elderly patients at increased mortality risk..[19-21] MTX usage is also associated with an increased risk of non-serious infections, including febrile neutropenia; however, by reducing the cumulative dose of GCs, it could paradoxically reduce this risk.[21,22] Similar considerations can be extended to the usage of anti-IL 6 drugs with observational studies observed an increased risk of lung infections.[16-22]

Here we present a case of *L. monocytogenes* brain abscess mimicking an ischemic stroke that occurred in a male patient with a previous diagnosis of PMR under immunosuppressive treatment. A literature revision was also performed. Written informed consent was obtained from the patient for publication of this case report.

Case Report

In March 2025, a 73-year-old Caucasian male was admitted to our Internal Medicine Unit due to the acute onset of left upper limb weakness, dysarthria, and mental confusion on the day before the admission. His medical history included parkinsonism, hypertensive heart disease with paroxysmal atrial fibrillation on direct anticoagulant drugs (DOAC), and benign prostatic hyperplasia. Eight months before the presentation, he received a diagnosis of PMR without GCA due to the presence of inflammatory pain in the scapular and pelvic girdle with wrist arthritis, increased inflammatory markers, and negative autoantibodies (*e.g.*, rheumatoid factor, anti-cyclic citrullinated peptides, and antinuclear antibodies).

A treatment with prednisone (PDN) and MTX was started in another rheumatological center. We do not know why it started MTX as first-line treatment because the patient was evaluated in another center, but we can speculate it was started due to the articular involvement as a steroid-sparing agent. He had a good response on articular manifestations and inflammatory markers.

On current hospital admission, he was regularly taking PDN 10 mg per day and MTX 15 mg subcutaneously every week with folic acid supplementation 24 hours after the MTX dose. Other chronic medications were rivaroxaban 20 mg/d, ramipril 5 mg/d, and carvedilol 2.5 mg/d.

In the context of pre-existing parkinsonism and chronic vascular burden, his Charlson Comorbidity Index was 6, and the Clinical Frailty Scale score was 6, indicating he was moderately frail.[23-25]

Physical examination revealed right upper limb hypertonia (spasticity) and left-sided hemi-inattention. Initial brain computed tomography (CT) suggested a left parietal ischemic lesion. Laboratory findings showed C-reactive protein 98 mg/L (normal value, n.v. 0-5), erythrocyte sedimentation rate 55 mm/h (n.v. 1-20), serum ferritin level 686 ng/mL (n.v. 21.8-274.6), serum iron 30 µg/dL (n.v. 65-175), and slight hypogammaglobulinemia 0.69 gr/dL (n.v. 0.80-1.35). Other laboratory tests were normal, including procalcitonin, leukocyte count with conserved differential, electrolytes, coagulation panel and hepato-renal function, metabolic profile, thyroid function tests, folic acid, cobalamin, homocysteine, Widal-Wright reaction, and anti-HIV 1/2 antibodies.

After the neurologist consultation, a diagnosis of ischemic stroke was made without indication for reperfusion due to the time lapse and the CT results. DOAC was temporarily stopped due to the risk of bleeding, and acetylsalicylic acid was started.

Despite initial management, after 2 days, the patient developed remittent fever, worsening of left-sided weakness evolving into left-sided hemiplegia, and a decline in consciousness becoming drowsy. A contrast-enhanced magnetic resonance imaging (MRI) of the brain showed a large abscess in the right parietal lobe with deep extension in the capsulo-thalamic region, compression of the lateral ventricle, and involvement of the corresponding leptomeningeal space.

Cerebrospinal fluid (CSF) analysis was clear with no leukocytes or erythrocytes, normal glucose, but hyperproteinorrachia 154.30 mg/dL (n.v. 15-45). An empirical antibiotic therapy with ceftriaxone 2 g every 12 hours and metronidazole 500 mg every 8 hours was started, with dexamethasone 4 mg every 12 hours and mannitol 18% in order to reduce the edema.

Cultures of blood and CSF resulted positive for *L. monocytogenes*. A transthoracic echocardiogram ruled out valvular vegetations, and a CT chest and abdomen showed no significant alterations. A final diagnosis of NL with brain abscess was established, and and intravenous (I.V.) ampicillin 2 g every 4 hours was started with progressive clinical improvement, but he was subsequently transferred to an infectious disease unit in another hospital and lost at follow-up. Unfortunately, we have no information regarding the continuation of the therapeutic process and the prognosis.

Discussion

We presented a case of NL with an atypical presentation mimicking an ischemic stroke, with a delayed onset of fever and infection signs, in a male patient with a previous diagnosis of PMR.

TTo the best of our knowledge, up to 31 December 2025, this is the first case of a brain abscess with deep extension in the capsulo-thalamic region, compression of the lateral ventricle, and involvement of the corresponding leptomeningeal space, caused by *L. monocytogenes* in a PMR patient treated with MTX and PDN. The only other case report reported in literature regarding an NL is about a male patient with PMR taking PDN with a native vertebral osteomyelitis and an epidural abscess.[26] NL includes 4 major clinical entities: meningitis, meningoencephalitis, rhombencefalitis, and brain abscess, and it may start with a stroke-like presentation. *L. monocytogenes* is the second agent responsible for acute bacterial meningitis after *Streptococcus pneumoniae* in immunocompromised patients. According to some epidemiological data, *L. monocytogenes* accounts for 16% of acquired bacterial meningitis in adults >80 years, up to 22% of brain infections in immunocompromised adults, 2-7% in neonates, and about 4% in young adults without risk factors.[3,27-30] Brain invasion generally occurs in the context of a disseminated disease with bacteremia, although the exact site of entry is not established. Probably *L. monocytogens* can survive in the gastrointestinal tract, traversing the intestinal barrier (both penetrating through the M-cells in the Peyer's patches or by the direct invasion of intestinal epithelium mediated by specific internalines) and gaining access to the reticulo-endothelial system in the spleen and liver and finally passing in the bloodstream (if the host immune response is not able to contain the infection). *L. monocytogenes* can enter cells both *via* the normal phagocytic function of macrophages and other antigen-presenting cells and different normally non-phagocytic cells such as enterocytes, fibroblasts, hepatocytes, or endothelial cells. After internalization, its PrfA-regulated virulence factors promote listerial intracellular survival. It uses intracellular metabolites and can spread directly from cytosol to cytosol, avoiding the exposure to the extracellular host defense systems (*e.g.*, antibodies, complement, and neutrophils). Infection removal depends on cytosolic innate immunity and the mounting of an efficacious cell-mediated immune response involving tumor necrosis factor-α (TNFα), interferon γ, M1

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(classically) activated macrophages, and major histocompatibility complex-class-Ia-restricted CD8+ T cells.[3]

L. monocytogenes can infect brain endothelial cells either directly or by cell-to-cell spreading from infected phagocytes entering into the CNS through the choroid plexus, ventricles, or brain microvasculature or *via* the trigeminal nerve by axonal spreading..[3,21,31,32]

Risk factors for NL include older age, cancer, diabetes mellitus, and use of GCs or other immunosuppressant drugs.[3] Recently, a study identified inflammatory bowel diseases as another risk factor, probably linked to the use of anti-TNFα drugs; proton pump inhibitor usage could facilitate the enterocyte infection by inhibiting acid secretion in the stomach.[33,34]

Patients with NL present with a slower onset of symptoms compared with other acute bacterial meningitis caused, for example, by meningococcus or pneumococcus. Although fever is frequently present (85-90% of cases), the classic bacterial meningitis triad of fever, neck stiffness and alteration of the mental status is less frequent, while 20% of patients develop a coma state and 10% present seizures.[3]

A CT scan is the first imaging modality used in the diagnostic work-up of a stroke-like syndrome due to its availability and short time of acquisition. Although it could reveal a macroscopic abscess, its sensitivity is low, in particular in the study of the posterior fossa or in meningoencephalitis, and, as in our case, in the early phase, it could not discriminate between stroke and brain abscess.[28] Contrast-enhanced CT can show the size and location of the abscess with a surrounding rim enhancement. However, MRI, using diffusion-weighted sequences and with the addition of the contrast medium, has a sensitivity and a specificity of about 90%, and it can distinguish between abscess and neoplasm.[28,33] Using spectroscopy MRI, it can be possible to distinguish abscesses from rim-enhancing tumors, and it can orientate towards the aetiology of the abscess according to the specific spectroscopic pattern found.[34]

As far as listeriosis in people with rheumatic diseases (RMDs) was concerned, in the literature, we found 73 case reports (Table 1) and 4 case series (Table 2), for a total of 396 patients. Median age was about 54 years (age was not specified in every study); 54.8% were females, the majority regarding patients with rheumatoid arthritis (RA) receiving immunosuppressive therapy (namely GCs, MTX, and anti-TNFα, among others) that presented predominantly with septic arthritis (mono-, oligo-, or polyarticular both in native and prosthetic joints).[35-46] One case was, curiously, associated with the use of acupuncture,[47] while another with intra-articular injection of Yttrium-90 therapy.[35] Other *L. monocytogenes* manifestations in RA patients included sepsis, meningitis, rombencephalitis, osteomyelitis, empyema, pericardial effusion and a splenic granuloma in a patient with Felty syndrome.[47-56]

Regarding other RMDs, there are cases in patients with juvenile idiopathic arthritis,[57] psoriatic arthritis, dermatomyositis, systemic lupus erythematosus, mixed connective tissue disease, GCA, sarcoidosis, and Still's disease.[39,58-82] Tables 1 and 2 reassume the main characteristics of RMD patients with *L. Monocytogenes* infection. It can be noted that MTX and GCs are frequently used by patients who developed infection, reflecting their central role and subsequent wide usage in the management of RMDs.

MTX is considered the anchor drug in the management of RA and as a steroid-sparing agent in many other RMDs, including PMR.[16,82] MTX has multiple immunomodulating effects, repressing T cell activation, downregulating B-cell function, and increasing the sensitivity of activated CD-95 T cells. Additionally, it inhibits the binding of IL-1 to its cell surface receptor. These effects may eventually favor *L. monocytogenes* infection and dissemination.[66,67] MTX is generally considered safe with regard to the infection risk; however, even low doses of MTX have been associated with *L. monocytogenes* infection.[68,69]

For many years, GCs have been considered another well-known risk factor for invasive listeriosis both in humans and in animal models with high morbidity and mortality.[70] They lead to alteration of cellular and humoral immunity and impair intracellular killing by membrane stabilization, facilitating the spreading of the infection. Moreover, it has to be said that many patients were also taking concomitant immunsupressive drugs boosting those effects.[70-82]

So in our case, it is reasonable to suppose that the use of GC, even if at low/medium dosage, associated with MTX, could have favored, in a cumulative manner, the infection and the subsequent brain invasion. It is interesting to note that the patient has a slight hypogammaglobulinemia probably induced by the immunosuppressive treatment. Unfortunately, we could not find a certain source of infection; in particular, the patient was denied consumption of raw milk products. Moreover, his age was higher than the median age of previous cases, and he also can be considered frail due to his comorbidities.

The uniqueness of this case also lies in its atypical presentation, which featured an acute, stroke-like onset. The subsequent appearance of fever and other infectious signs only 2 days after admission complicated clinical management, as initial efforts focused solely on the suspected cerebrovascular event, thereby delaying the initiation of empirical antibiotic therapy. Another abnormality is the absence of pleocytosis in CSF. In their paper, Horta-Baas *et al.* found the presence of liquor pleocytosis in all the cases they analyzed with a predominancy of polymorphonuclear cells.[69]

In this regard, it is plausible that the immunosuppression had blunted the immune response of the patient, slowing the appearance of the systemic response to the infection, thus mimicking an acute cerebrovascular accident and probably preventing the development of CSF pleocytosis. Moreover, a brain abscess could have a hematogenous origin with subsequent (asymptomatic bacteremia) contiguous involvement of leptomeninges, thus explaining the absence of CSF pleocytosis because there is first cerebritis rather than meningoencephalitis. It has to be said that CFS has been rapidly analyzed after the collection, so it is unlike that accidental degradation of white blood cells has occurred.

This, also, stresses the importance of obtaining early, pre-antibiotic, blood cultures in immunocompromised patients with a low threshold in order to isolate opportunistic pathogens and promptly institute an appropriate antimicrobic treatment.

Limitations of this study include a lack of follow-up due to the patient's transfer to another hospital in a different city, which precluded the assessment of the final clinical outcome; additionally, the definitive source of infection could not be identified. Regarding the latter, we do not have access to the results of the microbiological tests conducted by the public health office. However, establishing clear risk factors is frequently challenging in cases of listeriosis. *L. monocytogenes* can contaminate both processed and unprocessed food supply chains at multiple stages, and even low-level contamination can be clinically significant. Furthermore, potential vehicles of infection are numerous given the bacterium's remarkable environmental resilience. This epidemiological complexity is compounded by a variable incubation period, notably prolonged in cases involving pregnancy or CNS infections,[2] which further hinders retrospective source tracking.

Lastly, a stool culture was not performed, which could have provided valuable insights regarding gastrointestinal colonization by *L. monocytogenes*.

Table 1. Case reports of rheumatic disease patients with *Listeria monocytogenes* infection.

N.	Author[ref]	Sex, age	RMDs	Treatment	Type of listeriosis infection
1	Nadarajah *et al.*[60]	M, 63	RA	MTX, PRD, ETA	Septic arthritis, NJ
2	Wilson *et al*[34]	M, 70	RA	IA yttrium-90 injection	Septic arthritis, NJ
3	Inhorn *et al.*[56]	F, 48	RA/Felty's syndrome	GCs	Splenic granuloma
4	Booth *et al.*[35]	F, 64	RA	GCs, gold, AZA, PEN, PHEN	Septic arthritis, PJ
5	Rachapalli *et al.*[61]	M, 65	RA	ETA, PRD	Septic arthritis, NJ
6	Pagliano *et al.*[47]	M, 45	RA	ETA	Meningitis
7	Tweezer-Zaks *et al.*[48]	F, 55	RA	IFX, PRD, MTX	Sepsis
8	Murphy[49]	F, 58	RA	MTX, ADA, GCs	Sepsis
9	Gluck *et al.*[62]	F, 60	RA	IFX, MTX, PRD, CyA	Meningoencephalitis, sepsis
10	Gluck *et al.*[62]	F, 62	RA	MTX, CyA, IFX	Brain abscess
11	Tien *et al.*[46]	M, 78	RA	MTX, PRD, HCQ, COXIB,	Septic arthtritis NJ, after acupuncture
12	Turner *et al.*[56]	M, 19	JIA	High dose PRD, Colc	Meningitis, brainstem abscess
13	Benji *et al.*[50]	F, 87	RA	ETA, HCQ	Rhombencephalitis
14	Kesteman[38]	F, 52	MCTD	MTX, IFX, GCs	Sepsis, terminal ileitis
15	Kesteman[38]	M, 79	RA	MTX, IFX, GCs	Sepsis, NJ septic arthritis
16	Ukkonen *et al.* [36]	F, 73	RA	Gold, GCs, Antimalarials, SZ, PEN	Oligoarthtitis NJ
17	Huan-Ling *et al.*[57]	F, 47	DM	PRD	Meningitis
18	Huan-Ling *et al.*[57]	F, 56	SLE	PRD, CYC	Sepsis, MOF
19	Huan-Ling *et al.*[57]	F, 23	SLE	PRD	Acute liver failure hepatic encephalopathy, coma, GI bleed, respiratory failure
20	Huan-Ling *et al.*[57]	F, 33	SLE with nephropathy	PRD high dose	Sepsis
21	Huan-Ling *et al.*[57]	F, 43	DM	PRD, CYC	Sepsis, meningitis
22	Huan-Ling *et al.*[57]	F, 22	SLE with nephropathy	PRD, HCQ, DAP, MMF	Meningitis
23	Huan-Ling *et al.*[57]	M, 53	Still's disease	MTX, GCs	Meningitis
24	Huan-Ling *et al.*[57]	F, 18	SLE	None	Meningitis
25	Huan-Ling *et al.*[57]	M, 74	DM	None	Meningitis
26	Huan-Ling *et al.*[57]	F, 53	SLE with nephropathy	PRD, CYC	Meningitis
27	Huan-Ling *et al.*[57]	F, 45	MCTD	PRD	Meningitis
28	Huan-Ling *et al.*[57]	F, 49	DM	None	Sepsis
29	Huan-Ling *et al.*[57]	M, 59	DM	None	Meningitis
30	Crellin *et al.*[54]	F, 58	RA	PRD	Pericardial effusion
31	Venita Bowie *et al.*[51]	M, 73	RA	IFX, MTX, PRD	Meningitis
32	Georg Schett *et al.*[37]	F, 54	RA	ETA, MTX	Septic arthritis NJ
33	Peña-Sagredo *et al.*[44]	F, 63	PsA	IFX, MTX, PRD	Meningitis/sepsis
34	Peña-Sagredo *et al.*[44]	M, 57	PsA	IFX, MTX, PRD	Meningitis
35	Peña-Sagredo *et al.*[44]	F, 69	RA	ADA	Sepsis
36	Peña-Sagredo *et al.*[44]	F, 63	RA	IFX, MTX, PRD	Meningitis
37	Peña-Sagredo *et al.*[44]	F, 56	RA	IFX, MTX, PRD	Sepsis/Peritonitis
38	Peña-Sagredo *et al.*[44]	F, 36	RA	IFX, MTX, PRD	Endophalmitis
39	Zhang *et al.*[59]	F, 53	SLE	CYC, PRD	Multiple brain abscesses
40	Kubota *et al.*[52]	M, 73	RA	ADA, MTP, Iguratimod	Osteomyelitis
41	Sung Sim *et al.*[53]	F, 67	RA	PRD	Pleuric empyema
42	Shimizu *et al.*[28]	F, 79	GCA	PRD, MTX	Rhombencephalitis mimicking stroke
43	Ibironke *et al.*[26]	M, 60	PMR	PRD	Vertebral osteomyelitis and epidural abscess
44	Wollenhau *et al.*[66]	M, 70	RA	MTX	Septic arthritis PJ
45	Paranjape *et al.*[63]	M, 46	DM	MTX	Rhombencephalitis
46	Jansen *et al.*[64]	F, 17	SLE	MTX, PRD	Septic arthritis NJ
47	Newman *et al.*[39]	F, 64	RA, psoriasis	MTX	Septic arthritis NJ
48	Dixon *et al.*[40]	M, 47	RA	IFX	Meningitis
49	Dixon *et al.*[40]	M, 67	RA	ETA	Septic arthritis
50	Dixon *et al.*[40]	F, 60	RA	ADA	Septic arthritis

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Table 1. Continued from previous page.

N.	Author[ref]	Sex, age	RMDs	Treatment	Type of listeriosis infection
51	Horta-Baas *et al.*[69]	F, 47	SLE	MMF 3 gr/die+ PRD 0,5 mg/Kg/D	Cerebral abscess
52	Schulze *et al.*[70]	F, 19	SLE	None	Meningitis, bacteriemia
53	Bourn *et al.*[71]	F, 48	SLE	Steroids NS	Meningitis bacteriemia
54	Harisdangk *et al.*[72]	M, 31	SLE, chronic renal failure	PRD, nitrogen mustard	Meningitisbacteriemia
55	Kraus *et al.*[73]	F, 25	SLE	PRD (50 mg/d)	Meningitis
56	Kraus *et al.*[73]	F, 29	SLE, Fisher-Evans Syndrome	PRD 100 mg/d	Meningitis, bacteriemia
57	Kraus *et al.*[73]	F, 20	Inactive SLE, chronic renal failure	None	Meningitis
58	Kraus *et al.*[73]	F, 28	Inactive SLE	None	Meningitis
59	Kraus *et al.*[73]	F, 60	Inactive SLE	PRD 7,5 mg/d	Meningitis, bacteriemia
60	Kraus *et al.*[73]	M, 27	Active SLE	PRD 200 mg/d	Meningitis
61	Mylonakis *et al.*[74]	F, 16	SLE	Steroids, Azathioprine, MTX	Meningitis
62	Eckburg *et al.*[75]	F, 34	SLE, renal transplantation	Standard immunosuppression not specified	Brain abscess, bacteriemia
63	Eckburg *et al.*[75]	M, 50	Sarcoidosis	PRD 50	Brain abscess
64	Hung *et al.*[76]	F, 29	SLE	CYC+PRDN	Meningitis
65	Hung *et al.*[76]	F, 24	SLE	PRDN	Meningitis
66	Hung *et al.*[76]	F, 22	SLE	HCQ+PRDN	Meningitis, bacteriemia
67	Cone *et al.*[77]	F, 56	SLE	PRD	Brain abscess, bacteriemia
68	Baizabal-Carvallo *et al.*[78]	F, 38	SLE	None	Multiple brain abscesses, meningitis
69	Tobon *et al.*[79]	F, 18	SLE	AZA+PRD	Meningitis, bacteriemia
70	Tobon *et al.*[79]	F, 27	SLE, CRF in HD	MMF+PRD	Meningitis, bacteriemia
71	Tobon *et al.*[79]	F, 18	SLE	MMF+PRD	Meningitis, bacteriemia
72	Lee *et al.*[80]	F, 32	SLE	HCQ+MMF+MTP	Meningitis bacteriemia
73	Mc Caffrey *et al.*[81]	M, 57	SLE	PRD (20 mg/d)	Meningitis bacteriemia

ADA, adalimumab; AZA, azathioprine; CyA, cyclosporin-A; CYC, cyclophosphamide; Colc, colchicine; COXIB, COX-2 inhibithor; CRF, chronic renal failure; DAP, dapsone; DM, dermatomyositis; ETA, etanercept; GCs, glucorticoids; GCA, giant cell arteritis; HCQ, hydroxychloroquine; HD, hemodialysis; IA, intra-articular; IFX, Infliximab; JIA, juvenile idiopathic arthtritis; MCTD, mixed connective tissue disease; MMF, mycophenolate mofetil; MTP, metylprednisolone; MTX, methotrexate; NJ, native joint; NS, not specified; PEN, penicillamine; PHEN, phemylbutazone; PJ, prosthetic joint; PMR, polymyaglia rheumatica; PRD, prednisone; PRDN, prednisolone; PsA, psoriatic arhritis; RA, rheumatoid arthtritis; RMDs, rheumatic diseases; SLE, systemic lupus erythematosus; SZ, sulfasalazine.

Table 2. Case series of rheumatic disease patients with *Listeria monocytogenes* infection.

N	Author	N. patients; sex; median age	Types of diseases	DMARD	Types of infection
1	Salmon-Ceron *et al.*[41]	4; NS; NS	RA	Anti-TNFα NS	1 colitis+bacteriemia, 2 pneumonia+bacteraemia, 1 arthritis left shoulder
2	Bodro *et al.*[42]	266; 60Y; F 50,8%	RMD 127, IBD 101; CD 64; UC 25, hematologic disease 9; others 28	IFX 205; ETA 31; ADA 26; RTX 11; ABA 1; GOL 1	Listeriosis 116; meningitis or encephalitis 108; sepsis 42
3	Slifman *et al.*[43]	15; 69, 5Y; F 8, M 7	9 RA; 9 CD, 1 NR	14 IFX; 1 ETA; 7 MTX; 1 CYA; 2 AZA; 3 Mercaptopurine; 4 Mesalamine	1 septic arthritis, Sepsis/meningitis
4	Wallis *et al.*[45]	38; NS; NS	NS	IFX 36; ETA 2	NS

ABA, abatacept; ADA, adalimumab; Anti-TNFα, anti tumor necrosis factor-α; AZA, azathioprine; CD, Crohn disease; CYA, cyclosporin-A; DMARD, disease modifung anti-rheumatic drug; ETA, etanercept; GOL, golimumab; IBD, inflammatory bowel disease; IFX, infliximab; MTX, methotrexate; NS, not specified; NR, not reported; RA, rheumatoid arthritis; RMD, rheumatic disease; RTX, rituximab; UC, ulcerative colitis.

Conclusions

Although stroke-like appearance is a rare, well-know manifestation on NL,[3,26,27] this is the first case in a patient with an RMD, specifically PMR. We reported the case of a patient with PMR treated with GCs and MTX who, insidiously, developed a cerebral abscess with involvement of leptomeningeal space due to *L. monocytogenes* mimicking an acute ischemic stroke. Immunosuppression, even low doses, long-term GCs, and MTX represented a risk factor for the infection. Moreover, this case report underlies the atypical presentation that NL can have in immunocompromised patients mimicking cerebrovascular accidents and delaying the correct diagnosis even with confounding laboratory tests. A high index of suspicion is needed, and microbiological samples must be obtained in order to promptly start adequate antimicrobial therapy and improve survival.

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