ANNALES ACADEMIAE MEDICAE SILESIENSIS

ACCEPTED MANUSCRIPT

Ann. Acad. Med. Siles. (Online) 2026; DOI: 10.18794/aams/216309

Case report

The listeriosis brain abscess – the extremely rare complication during immunosuppressive therapy of autoimmune hepatitis

Karolina Osińska[1], Joanna Musialik[1,2], Agnieszka Morawa[1]

[1]Department of Nephrology, Transplantation and Internal Medicine, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland
[2]Department of Gastroenterology and Hepatology, Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland

Address for correspondence:
dr hab. n. med. Joanna Musialik
ul. Medyków 14, 40-752 Katowice
Uniwersyteckie Centrum Kliniczne
im. prof. K. Gibińskiego ŚUM
e-mail: jmusialik@sum.edu.pl

Received: 26.11.2025, Revised: 10.12.2025, Accepted: 31.12.2025, Published: April 2026

Publisher: Medical University of Silesia, Katowice, Poland

ABSTRACT

In recent years, there has been a steady increase in the number of people receiving immunosuppressive treatment. The ongoing development of these therapies, although effective in controlling autoimmune disease, brings with it new diagnostic and therapeutic challenges due to complex modulation of cellular and humoral responses. Despite significant therapeutic benefits, their use is associated with the risk of developing opportunistic infections, which are almost unrecognised in immunocompetent persons. We present a case of neuroinfection caused by Listeria monocytogenes complicated by brain abscess in an autoimmune hepatitis female patient treated with prednisone and mercaptopurine. Importantly, the disease initially presented with non-specific neurological symptoms, which might have delayed diagnosis. Nevertheless, an early lumbar puncture, cerebrospinal fluid testing with identification of the most common pathogens, and imaging tests allowed for diagnosis and implementation of appropriate antibiotic therapy, which resulted in the expected clinical improvement. This case highlights that patients receiving immunosuppressive therapy may develop serious opportunistic infections, and early diagnosis and implementation of right treatment are crucial for improving prognosis and minimising complications.

KEYWORDS

autoimmune hepatitis, immunosuppressive treatment, listeriosis, brain abscess

INTRODUCTION

During the last decades the number of autoimmune disorders has increased and immunosuppressive therapy, the only therapeutic option, is used wider and wider [1]. This complex therapy suppresses the excessive immune response and reduces inflammation, which are the main causes of tissue damage in these conditions [2]. But immunosuppressive therapy is a double-edged sword – except for controlling the disease it carries many consequences and complications for the patient. One of them is increased susceptibility to infections that are practically unseen in immunocompetent patients [1]. We present a case with an extremely rare complication of immunosuppressive therapy in an autoimmune hepatitis patient.

CASE REPORT

A 64-year-old woman with autoimmune hepatitis diagnosed six months earlier and treated with 10mg of prednisone and mercaptopurine (50mg once a day) was urgently admitted to the emergency unit because of high blood pressure (180/100mmHg) and severe headache. For 24-hours

the patient suffered from headache, for a few hours accompanied by disorientation, lethargy and general weakness. A similar, albeit less severe, episode of disturbance in the general state, including slowdown, increased drowsiness and headache, had occurred about two weeks earlier but after three days the symptoms had gone away spontaneously, and the patient had not consulted her doctor. Actually, on admission, the neurological examination showed psychomotor retardation with no meningeal symptoms, and no focal signs of nervous system damage but the Romberg test was shaky, and the patient's gait was slowed, requiring assistance. An initial laboratory investigation (Tab. I) suggested an inflammatory process (high C-reactive protein and leucocytosis), and, despite the normal brain CT-scan the consultant neurologist decided to perform a lumbar puncture. In the cerebrospinal fluid (CSF) the increased protein concentration (1.85 g/l; N:0.15-0.45 g/l), and cytosis (726/µl; N:<5/µl) were found with the normal CSF glucose level (54 mg/dl, approx. 60% of the serum glucose level 50-80%) so a diagnostic panel for nervous system infections was performed including bacteria (Escherichia coli, Haemophilus influenzae, Listeria monocytogenes, Neisseria meningitidis, Streptococcus agalactiae, Streptococcus pneumoniae), viruses (cytomegalovirus, enterovirus, human herpesvirus types 1, 2, 6, human parvovirus, varicella zoster virus) and fungi (Cryptococcus neoformans). As a result, the infection with Listeria monocytogenes was diagnosed and confirmed by the CSF culture. For a few days, despite the inclusion of stepwise antibiotic therapy, the expected improvement in health was not obtained. The Magnetic resonance (MR) examination of the head on the 6th day of hospitalization showed the appearance of an inflammatory-purulent process in the brain with presence of brain abscess (Fig. 1A–B). Intensification of therapy, combined ampicillin and sulfamethoxazole with trimethoprim, resulted in a gradual improvement of the condition, and MR repeated on the 20th day of hospitalization revealed regression of the inflammatory changes described earlier (Fig. 2A–B). After 17 days of ampicillin and 4 days of sulfamethoxazole and trimethoprim treatment, the used antibiotics were replaced by linezolid - due to allergic reaction to the previous antibacterial treatment. With the gradual improvement in clinical and radiological conditions the patient was discharged on the 29th day of therapy. To date, one year after the disease, the patient is currently in stable condition with a slight memory impairment and remains under outpatient neurological follow-up.

Table I. Laboratory findings during the diagnostic and therapeutic process

Parameter	Reference range	Admission	2 weeks of therapy	Discharge	Three months after discharge
WBC[x10³/µ]	4-10	16,37	10,33	10,28	8,9

CRP [mg/l]	<5	62,9	2,3	-	1,3
ALT [U/l]	<35	76	70	63	21
AST [U/l]	<35	37	25	29	25
GGTP [U/l]	<35	75	221	137	41
INR	0,80-1,20	1,47	-	-	1,0
Creatinine [mg/dl]	0,7-1,1	0,8	0,7	-	0,74
Bilirubin [mg/dl]	0,3-1,2	2,1	-	-	1,07

WBC - white blood cells; CRP - C-reactive protein; ALT - alanine aminotransferase; AST - aspartate aminotransferase; GGTP - gamma-glutamyl transpeptidase; INR - international normalised ratio

DISCUSSION

Autoimmune hepatitis (AIH), like other autoimmunological diseases, is a disease with a complex pathogenesis with both genetic, immunological factors and environmental triggers, which are postulated in the disease development, but their significance is of uncertain value [3]. As with diseases of such nature, treatment includes immunosuppressive drugs, whose aim is to suppress the excessive immune response and reduce inflammation, which are the main causes of tissue damage in these conditions [2]. Its purpose is to achieve a clinical remission and a complete reversal at a biochemical and histological level [3,4]. The currently recommended treatment for AIH patients is immunosuppressive combination with glucocorticosteroid and azathiophrine/mercaptopurine (thiopurine derivatives). Their influence over the immune system involves several mechanisms, including inhibition of T and B lymphocyte proliferation, decreased cytokine production, impaired activity of phagocytic cells and disruption of both opsonization and angiogenesis [5]. The impairment in T cell mediated immunity seems to be crucial for opportunistic infections with the estimated risk even 136-fold higher in steroid treated patients with rheumatoid disease [6].

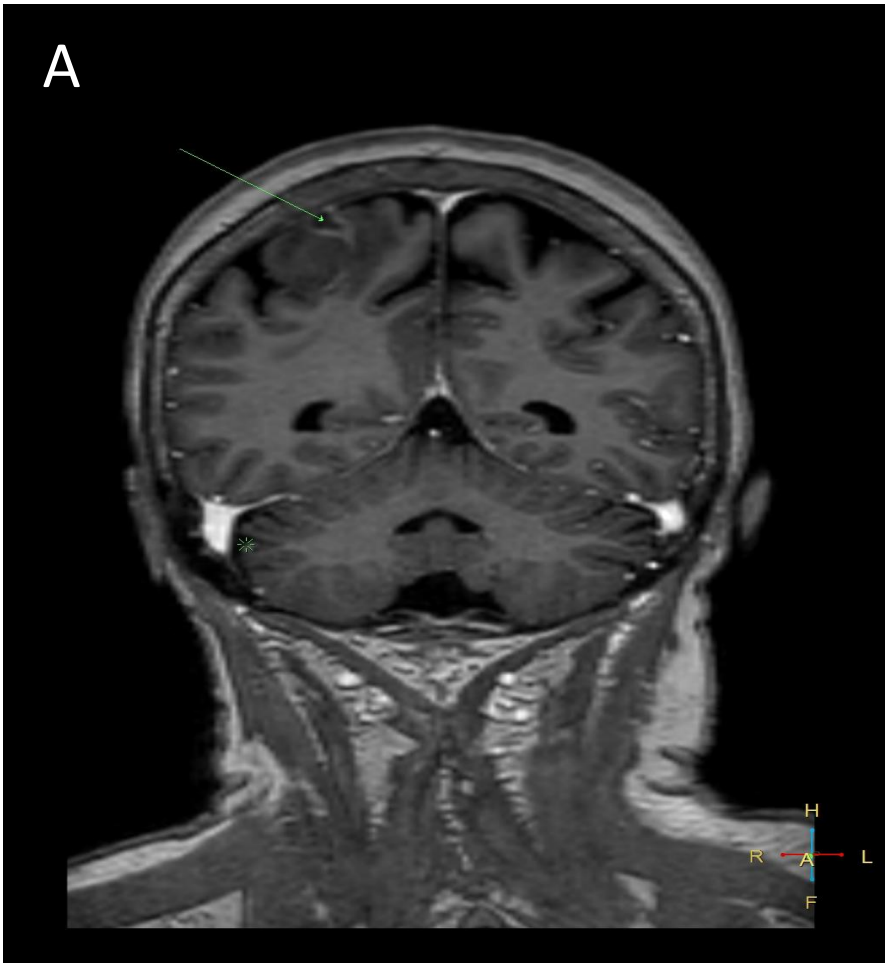
Commonly observed infections include viral diseases caused by cytomegalovirus or herpes simplex virus, fungal infections such as candidiasis and aspergillosis and bacterial infections, including the reactivation of latent tuberculosis [2]. In a study by Naganuma et al. [7], opportunistic infections in 9.1% of inflammatory bowel diseases patients were developed. The analysis showed that thiopurine derivatives use was an independent risk factor for these infections. Among the pathogens identified, herpes simplex virus (HSV), chickenpox (Varicella zoster virus) were the most common. Immunosuppression has been also identified as an independent risk factor for Clostrididoides difficile infection and is also associated with higher risk of recurrence [8]. It is worth noting that immunocompromised patients may also be affected by less common pathogens which can complicate both diagnosis and treatment.

Listeria monocytogenes (LM) is a Gram-positive, facultatively anaerobic bacterium. Despite its inability to form spores, it is widespread in the environment due to its minimal nutritional requirements and its ability to grow within a wide range of temperatures and environments containing up to 10% salt. The bacterium is a facultative intracellular pathogen, capable of crossing physiological barriers, including the intestinal, placental and blood-brain barriers. It can replicate inside macrophages and invade various types of non-phagocytic cells, spreading directly from cell to cell [9]. Humans typically acquire LM through consumption of improperly handled or stored food. Common sources include milk and dairy products, soft and mold-ripened cheeses, raw meat and vegetables, seafood, frozen foods, ready-to-eat delicatessen items, and occasionally vacuum-packed cold cuts. Although this bacterium is widespread in the environment, listeriosis remains uncommon and primarily affects individuals belonging to high-risk groups. Moreover, most infections in healthy individuals are asymptomatic or manifest as mild febrile gastroenteritis [10]. The most susceptible groups to develop severe infection include pregnant women, foetuses, newborns, older adults (particularly those over 50 years of age) and persons with impaired immune function. This group encompasses patients suffering from malignant tumours, diabetes or other chronic diseases, as well as those undergoing immunosuppressive treatment or organ transplantation [11]. In high-risk individuals LM can invade the central nervous system, leading to severe neurological manifestations. Clinical symptoms are often nonspecific and include fever, headache, vomiting, and altered consciousness [12]. This facultative intracellular bacterium exhibits a particular tropism for the nervous system, causing meningitis, encephalitis and inflammation of the cerebellum and brainstem (rhombencephalitis). The mechanisms, by which LM reaches the brain, are not fully understood, but in humans, the bloodstream is most often suggested as the main route of invasion [13].

In literature, there are very few cases of neuroinfection caused by LM. Zhang et al. [14] described a case of a 64-year-old female patient suffering from nephrotic syndrome treated with high doses of glucocorticosteroids with cyclophosphamide, which contributed to an intracranial infection with

lesions in the fronto-parietal region of the brain. In further investigation revealed that the lesions were caused by Listeria monocytogenes. The case study by Vasconcelos et al. [11] concerned a 77-year-old man treated with glucocorticosteroids for bronchiolitis obliterans organizing pneumonia. The medical history revealed supratentorial neuroinfection. MR examination showed a left frontal subcortical mass lesion. The case required surgical debridement, during which purulent material was collected. Listeria monocytogenes was isolated in all purulent material, CSF and blood as well. Bristowe et al. [15] reported another case of listerial neuroinfection. In an 82-year-old male patient the lesions were located in the right frontal lobe of the brain. The patient did not take immunosuppressive drugs, but the predisposing factor was confirmed type 2 diabetes.
Up to our best knowledge, only two cases of autoimmune hepatitis and neurolisteriosis have been described in medical literature so far. Trachuk et al. [16] described the case of a 56-year-old female AIH patient treated with prednisone and azathioprine and LM infection complicated with the abscess formation in the left frontoparietal region of the brain. Except targeted treatment with ampicillin and gentamicin, the drainage of the abscess was necessary. Due to the medical interventions, the patient achieved significant improvement, however, minor neurological deficits remained. Ezquerra et al. [17] published a case study of a 71-year-old man diagnosed with liver cirrhosis secondary to AIH treated with methylprednisolone alone. The infection of LM led to meningitis and an abscess formation. The recommended therapy with ampicillin and gentamicin led to successful cure and symptoms and signs recovery.

The aim of this review was to present a rare but extremely serious complication of immunosuppressive therapy – neuroinfection with brain abscess caused by a rare pathogen. The signs and symptoms of the disease were nonspecific and developed slowly. We would like to pay attention to the fact that immunosuppressive, biological or systemic chemo- therapy could lead to unexpected infectious complications and we should have exceptional vigilance in care of these patients.



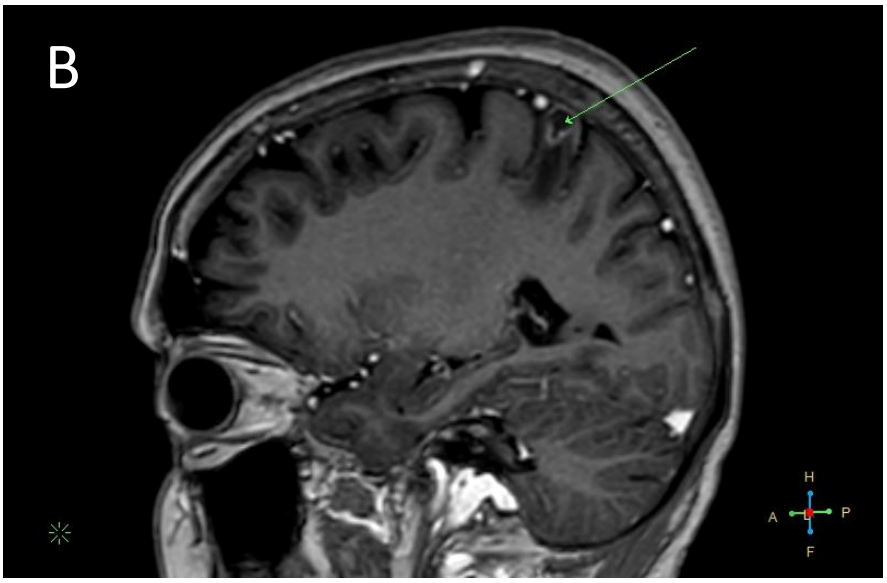


Fig. 1A, B. A quite extensive zone of oedema penetrating towards the white matter of the coronary radiata in the right brain hemisphere with a diameter of 25 mm (arrow). Within this area, subcortically by the parietal scale there was an annular plate area with a size of 10 mm with a strongly strengthening rim.

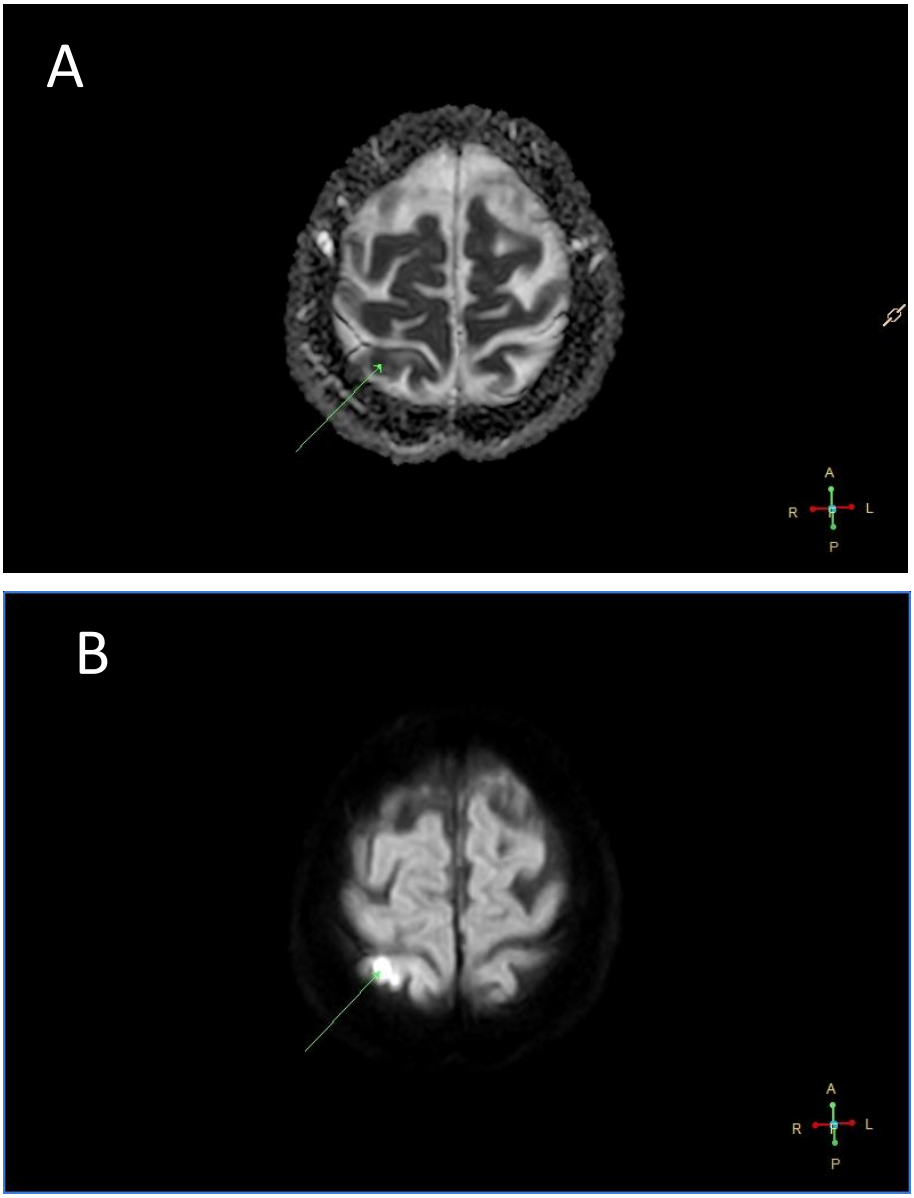


Fig. 2A, B. A significant regression of oedematous changes in the right parietal lobe, as well as in an area of pathological contrast enhancement, which is currently 7 mm in diameter (arrow), with the appearance of a purulent lesion in the regression phase.

Authors' contribution

Study design - J. Musialik

Data collection - J. Musialik

Manuscript preparation - K. Osińska, A. Morawa, J. Musialik

Literature research - K. Osińska, A. Morawa, J. Musialik

Final approval of the version to be published - J. Musialik

Acknowledgements

We would like to thank Dr Anna Kwaśniewska, MD, PhD for her help in magnetic resonance scans analysis and prepare for publication.

REFERENCES

1. Giles AJ, Hutchinson MND, Sonnemann HM, Jung J, Fecci PE, Ratnam NM, et al. Dexamethasone-induced immunosuppression: mechanisms and implications for immunotherapy. J Immunother Cancer. 2018;6(1):51. doi: 10.1186/s40425-018-0371-5.
2. Orlicka K, Barnes E, Culver EL. Prevention of infection caused by immunosuppressive drugs in gastroenterology. Ther Adv Chronic Dis. 2013;4(4):167–185. doi: 10.1177/2040622313485275.
3. Manns MP, Vogel A. Autoimmune hepatitis, from mechanisms to therapy. Hepatology. 2006;43(2 Suppl 1):132–144. doi: 10.1002/hep.21059.
4. Czaja AJ, Freese DK; American Association for the Study of Liver Disease. Diagnosis and treatment of autoimmune hepatitis. Hepatology. 2002;36(2):479–497. doi: 10.1053/jhep.2002.34944.
5. Yassin W, Nasser R, Veitsman E, Saadi T. The Effectiveness of Measuring Thiopurine Metabolites in the Treatment of Autoimmune Hepatitis Patients. Turk J Gastroenterol. 2024;35(3):232–238. doi: 10.5152/tjg.2024.22838.
6. Cutolo M, Seriolo B, Pizzorni C, Secchi ME, Soldano S, Paolino S, et al. Use of glucocorticoids and risk of infections. Autoimmun Rev. 2008;8(2):153–155. doi: 10.1016/j.autrev.2008.07.010.
7. Naganuma M, Kunisaki R, Yoshimura N, Takeuchi Y, Watanabe M. A prospective analysis of the incidence of and risk factors for opportunistic infections in patients with inflammatory bowel disease. J Gastroenterol. 2013;48(5):595–600. doi: 10.1007/s00535-012-0686-9.
8. Lübbert C, Johann C, Kekulé AS, Worlitzsch D, Weis S, Mössner J, et al. Immunosuppressive treatment as a risk factor for the occurrence of clostridium difficile infection (CDI). [Article in German]. Z Gastroenterol. 2013;51(11):1251–1258. doi: 10.1055/s-0033-1335505.
9. Wałecka-Zacharska E, Bania J. *Listeria monocytogenes* – patogen, który wie, jak przetrwać. Życie Wet. 2014;89(11):917–919.
10. Godziszewska S, Musioł E, Duda I. Listeriosis – a dangerous, contagious disease. Meningitis caused by Listeria monocytogenes – case report. [Article in Polish]. Ann Acad Med Siles. 2015;69:118–124. doi: 10.18794/aams/33100.
11. Vasconcelos M, Moreira AP, Pereira CS, Duarte Armindo R, Noronha C. Brain Abscess Caused by Listeria monocytogenes: A Rare Case of Supratentorial Neurolisteriosis. Cureus. 2024;16(2):e54521. doi: 10.7759/cureus.54521.
12. Qu H, Wang Y, Diao H, Ren G, Wang Z, Shang J, et al. Clinical characteristics of 15 patients with Listeria meningitis in adult. Heliyon. 2023;10(1):e23755. doi: 10.1016/j.heliyon.2023.e23755.

13. Disson O, Lecuit M. Targeting of the central nervous system by Listeria monocytogenes. Virulence. 2012;3(2):213–221. doi: 10.4161/viru.19586.
14. Zhang J, Huang S, Xu L, Tao M, Zhao Y, Liang Z. Brain abscess due to listeria monocytogenes: A case report and literature review. Medicine. 2021;100(31):e26839. doi: 10.1097/MD.0000000000026839.
15. Bristowe H, Dissanayake K, Chandra J, Arias M. Listeria brain abscess: a therapeutically challenging rare presentation of listeriosis. BMC Infect Dis. 2024;24(1):477. doi: 10.1186/s12879-024-09295-z.
16. Trachuk P, Marin Saquicela T, Levi M, Khedimi R. Listeria brain abscess in a patient with autoimmune hepatitis. IDCases. 2019;17:e00569. doi: 10.1016/j.idcr.2019.e00569.
17. Ezquerra A, Martínez B, García-Buey L. Invasive listeriosis in a patient with autoimmune hepatitis on glucocorticosteroid therapy. Med Clin. 2022;158(1):39. doi: 10.1016/j.medcli.2021.03.015.