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OPIS PRZYPADKU
CASE REPORT

Listeriosis brain abscess: An extremely rare complication during immunosuppressive therapy for autoimmune hepatitis

Ropień mózgu w przebiegu neuroinfekcji wywołanej przez *Listeria monocytogenes* – niezwykle rzadkie powikłanie w trakcie leczenia immunosupresyjnego autoimmunologicznego zapalenia wątroby

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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 diseases, brings with it new diagnostic and therapeutic challenges due to complex modulation of cellular and humoral responses. Despite significant therapeutic benefits, their use entails the risk of developing opportunistic infections, which are almost unrecognized in immunocompetent persons. We present a case of neuroinfection caused by *Listeria monocytogenes* that was complicated by a brain abscess in a female autoimmune hepatitis patient being treated with prednisone and mercaptopurine. Importantly, the disease initially presented with non-specific neurological symptoms, which might have delayed the diagnosis. Nevertheless, an early lumbar puncture with cerebrospinal fluid testing to determine the presence of the most common pathogens and imaging tests led to a diagnosis and appropriate antibiotic therapy, which resulted in the expected clinical improvement. This case highlights how patients receiving immunosuppressive therapy may develop serious opportunistic infections and that early diagnosis and the right treatment are crucial for improving prognosis and minimizing complications.

KEYWORDS

autoimmune hepatitis, immunosuppressive treatment, listeriosis, brain abscess

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STRESZCZENIE

W ostatnich latach obserwuje się systematyczny wzrost liczby chorych, u których stosowane są terapie modulujące działanie układu odpornościowego. Oprócz niewątpliwej skuteczności w leczeniu chorób autoimmunologicznych, leczenie to niesie za sobą wyzwania związane ze zwiększonym ryzykiem rozwoju zakażeń, w tym oportunistycznych, które praktycznie nie są rozpoznawane u osób z prawidłowo funkcjonującym układem immunologicznym. W pracy przedstawiono przypadek neuroinfekcji *Listeria monocytogenes* powikłanej ropniem mózgu u 64-letniej chorej, leczonej od kilku miesięcy prednizonem i merkaptopuryną z powodu autoimmunologicznego zapalenia wątroby, przebiegającej początkowo z niespecyficznymi objawami neurologicznymi. Punkcja lędźwiowa z oceną płynu mózgowo-rdzeniowego, obejmująca identyfikację najczęstszych patogenów, pozwoliła na ustalenie rozpoznania. Prezentowany przypadek zwraca uwagę na poważne infekcje o nietypowym przebiegu klinicznym u chorych leczonych immunosupresyjnie, a wczesna diagnostyka i wdrożenie odpowiedniej terapii mają kluczowe znaczenie dla poprawy rokowania i minimalizacji powikłań.

SŁOWA KLUCZOWE

autoimmunologiczne zapalenie wątroby, leczenie immunosupresyjne, listerioza, ropień mózgu

INTRODUCTION

In recent decades, the number of autoimmune disorders has increased and the only therapeutic option for them, immunosuppressive therapy, is being used more and more [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: apart from controlling the disease, it can bring about many consequences and complications for the patient. One of them is increased susceptibility to infections which are practically unseen in immunocompetent patients [1]. We present a case with an extremely rare complication of immunosuppressive therapy in an autoimmune hepatitis (AIH) patient.

CASE REPORT

A 64-year-old woman with AIH, diagnosed six months earlier, who was being treated with 10 mg of prednisone and mercaptopurine (50 mg once a day) was urgently admitted to the emergency unit because of high blood pressure (180/100 mmHg) and severe headache. For 24 hours, the patient had suffered from headache; for a few hours, it had been accompanied by disorientation, lethargy, and general weakness. A similar, albeit less severe, episode of disturbance in her general state – including slowdown, increased drowsiness, and headache – had occurred about two weeks earlier, but the symptoms had gone away spontaneously after three days and the patient had not consulted her doctor. 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 slow and she required assistance. An initial laboratory investigation (Table I) suggested an inflammatory process (high C-reactive protein and leucocytosis) and the consultant neurologist decided to perform a lumbar puncture despite a brain computed tomography scan being normal. In the cerebrospinal fluid (CSF), elevated protein concentration (1.85 g/l; N: 0.15–0.45 g/l) and cytosis (726/µl; N: <5/µl) were found; the CSF glucose level was normal (54 mg/dl, approx. 60% of the serum glucose level 50%–80%). Therefore, a diagnostic panel for nervous system infections was performed, including bacteria (*Escherichia coli*, *Haemophilus influenzae*, *Listeria monocytogenes*, *Neisseria meningitidis*, *Streptococcus agalactiae*, and *Streptococcus pneumoniae*), viruses (cytomegalovirus, enterovirus, human herpesvirus types 1, 2, and 6, human parvovirus, and varicella zoster virus), and fungi (*Cryptococcus neoformans*). As a result, infection with *Listeria monocytogenes* (LM) was diagnosed and confirmed by CSF culturing. For a few days, despite the initiation of stepwise antibiotic therapy, the expected improvement in health was not observed. Magnetic resonance imaging (MRI) of the head on the 6th day of hospitalization showed an inflammatory-purulent process in the brain and a brain abscess (Figure 1A–B). The therapy was intensified (combined ampicillin and sulfamethoxazole with trimethoprim), resulting in a gradual improvement of the patient's condition, and an MRI scan repeated on the 20th day of hospitalization revealed a regression of the inflammatory changes (Figure 2A–B). After 17 days of ampicillin and 4 days of sulfamethoxazole and trimethoprim treatment, an allergic reaction to the antibacterial treatment led to the antibiotics being replaced by linezolid. Due to

a gradual improvement in the clinical and radiological conditions, the patient was discharged on the 29th day of therapy. One year after hospitalization, the patient is in stable condition with slight memory impairment; she remains under outpatient neurological follow-up care.

Table I. Laboratory findings during the diagnostic and therapeutic process

Parameter	Reference range	Admission	After two weeks of therapy	At discharge	Three months after discharge
WBC [×10³/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
5 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 normalized ratio

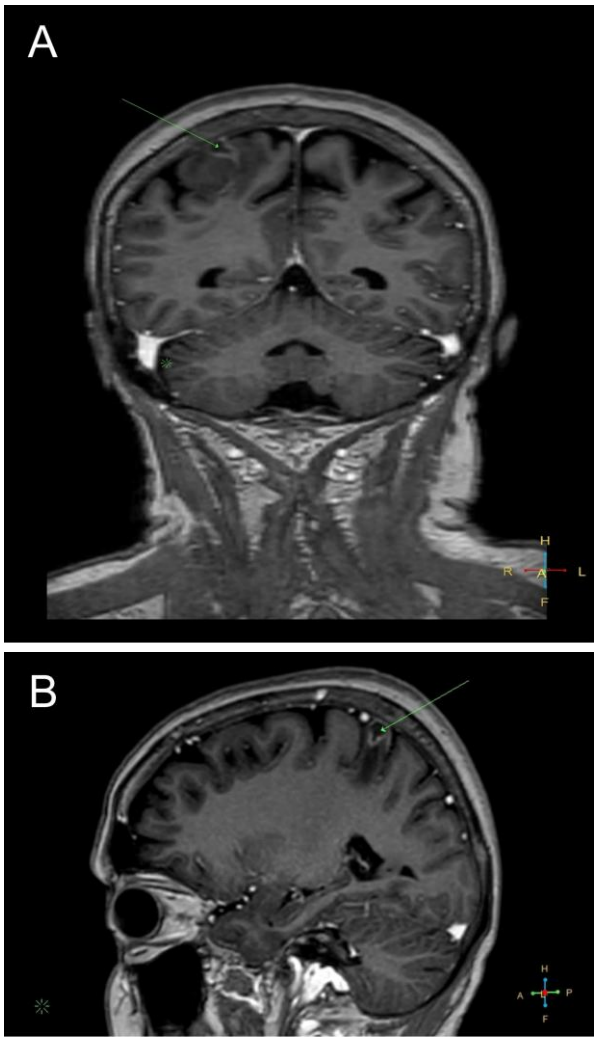

Fig. 1A–B. A quite extensive zone of edema penetrating toward the white matter of the coronary radiata in the right brain hemisphere with a diameter of 25 mm (arrow). Within this area, subcortically by the parietal scale, there was an annular plate area 10 mm in size with a strongly strengthening rim

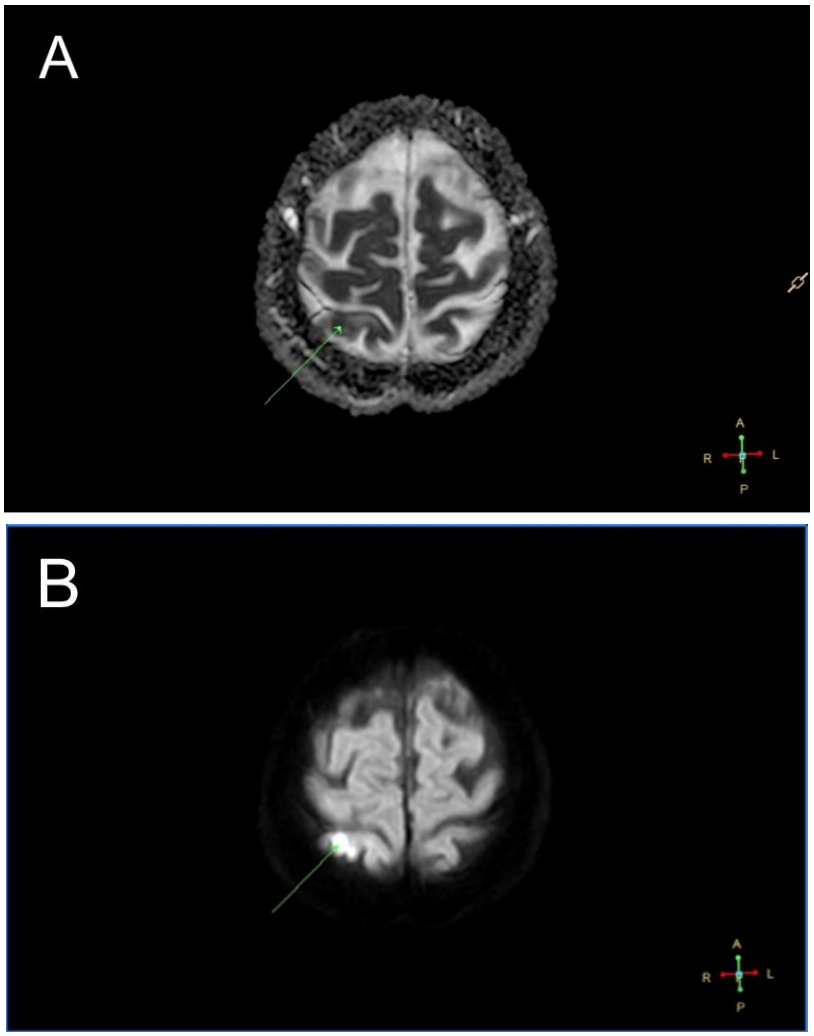

Fig. 2A–B. A significant regression of edematous changes in the right parietal lobe and in an area of pathological contrast enhancement, which is currently 7 mm in diameter (arrow), with the appearance of a purulent lesion in the regression phase

DISCUSSION

As with other autoimmune diseases, AIH has a complex pathogenesis, with both genetic and immunological factors as well as environmental triggers being postulated in the disease's development, but of uncertain significance [3]. Treatment for such diseases includes immunosuppressive drugs, the aim of which is to suppress the excessive immune response and reduce inflammation – the main causes of tissue damage in these conditions [2]. Its purpose is to achieve clinical remission and a complete reversal at the biochemical and histological level [3,4]. The currently recommended treatment for AIH patients is a combination of immunosuppressive drugs: glucocorticosteroids 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, as the estimated risk is 136-fold higher in steroid-treated patients with rheumatic diseases [6]. Commonly observed infections include viral diseases caused by cytomegalovirus or herpes simplex virus (HSV), 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 developed in 9.1% of inflammatory bowel disease patients. The analysis showed that the use of thiopurine derivatives was an independent risk factor for these infections. HSV and chickenpox (varicella zoster virus) were the most commonly identified pathogens. Immunosuppression has also been identified as an independent risk factor for *Clostridioides difficile* infection and it is associated with a 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.

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 infection by consuming improperly handled or stored food. Common sources include milk and dairy products, soft and mold-ripened cheeses, raw meat, raw vegetables, seafood, frozen foods, ready-to-eat foods, and occasionally vacuum-packed cold cuts. Although this bacterium is widespread in the environment, listeriosis remains uncommon and primarily affects individuals in high-risk groups. Moreover, most infections in healthy individuals are asymptomatic or manifest as mild febrile gastroenteritis [10]. The groups most susceptible to developing a severe infection are pregnant women, fetuses, newborns, older adults (particularly those over 50 years of age), and persons with impaired immune function. This last group encompasses patients suffering from malignant tumors, 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 the bloodstream is most often suggested as the main route of invasion in humans [13].

In the literature, there are very few cases of neuroinfection caused by LM. Zhang et al. [14] described the case of a 64-year-old female patient suffering from nephrotic syndrome and being treated with high doses of glucocorticosteroids and cyclophosphamide, which contributed to an intracranial infection with lesions in the frontoparietal region of the brain. Further investigation revealed that the lesions were caused by LM. A 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. MRI showed a left frontal subcortical mass lesion. The case required surgical debridement, during which purulent material was collected. LM was isolated in all purulent material, as well as the CSF and blood. Bristowe et al. [15] reported another case of LM neuroinfection. In an 82-year-old male patient, the lesions were located in the right frontal lobe of the brain. The patient was not taking immunosuppressive drugs, but the predisposing factor was confirmed type 2 diabetes.

To the best of our knowledge, only two cases of AIH and neurolisteriosis have been described in the medical literature so far. Trachuk et al. [16] described the case of a 56-year-old female AIH patient treated with prednisone and azathioprine for LM infection complicated with an abscess in the left frontoparietal region of the brain. Targeted treatment with ampicillin and gentamicin was administered and the abscess was drained. Due to these medical interventions, the patient achieved significant improvement; however, minor neurological deficits remained. Ezquerra et al. [17] published the case study of a 71-year-old man diagnosed with liver cirrhosis secondary to AIH who was treated with methylprednisolone alone. The LM infection led to meningitis and an abscess. The recommended therapy with ampicillin and gentamicin led to a reduction of the symptoms and signs of 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 in this case study were nonspecific and developed slowly. We would like to draw attention to the fact that immunosuppressive, biological, or systemic chemotherapy can lead to unexpected infectious complications and to encourage clinicians to be exceptionally vigilant when caring for these patients.

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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, K. Osińska, A. Morawa

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