CASE REPORT

LISTERIA MONOCYTOGENES BRAIN ABSCESS: TWO CASES AND REVIEW OF THE LITERATURE

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Abstract. *Listeria monocytogenes* is a gram-positive bacillus that exhibits predilection to infect the central nervous system in immunocompromised individuals; the most common manifestations are meningitis and rhombencephalitis. Listerial brain abscesses are rare. We report here two brain abscess cases caused by *L. monocytogenes* in patients receiving immunosuppressive agents. The first patient presented with left hemiparesis mimicking stroke and the second patient presented with neurological symptoms without fever, which was indistinguishable from brain tumor. In both cases, magnetic resonance spectroscopy (MRS) was performed to differentiate infectious processes from other causes. Diagnosis was made with a positive blood culture in both cases. Listerial DNA was detected in the pus aspirated from the abscess in the first case. Both patients were successfully treated with intravenous ampicillin followed by oral amoxicillin. MRS was useful in differentiating infectious processes from non-infectious causes.

Keywords: *Listeria monocytogenes*, listeriosis, brain abscess, listerial brain abscess

INTRODUCTION

Listeria monocytogenes is a non-spore forming non-branching gram-positive bacillus, which can cause serious infections in immunocompromised patients, especially in those with a malignancy or using corticosteroid. Common clinical manifestations of listeriosis include meningitis and rhombencephalitis (Armstrong and Fung, 1993). In rare occasions, this bacterium can cause infections in the parenchyma of the cerebral cortex, resulting in a brain abscess. Ampicillin and trimethoprim/sulfamethoxazole can be used to treat listerial brain abscesses. We report two patients with listerial brain abscesses whose presentations mimicked stroke and brain tumor.

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CASE REPORT

Case 1

A 68-year-old female presented to our hospital with low grade fever, severe headache and progressive left-sided weakness 1 week following the second course of chemotherapy which was commenced 5 weeks earlier to treat breast

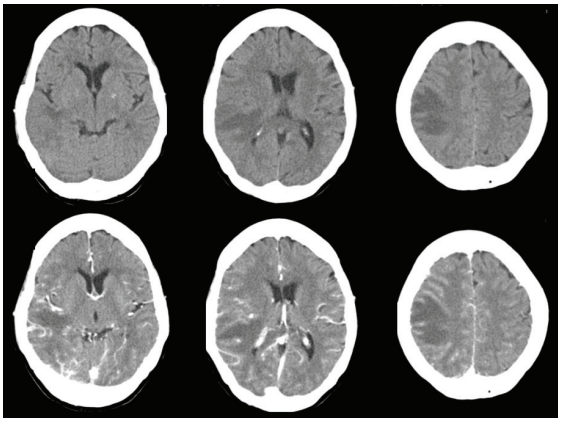

Fig 1–CT brain with contrast media of the first patient showing a hypodense lesion without abnormal enhancement in the right frontotemporoparietal lobe with surrounding vasogenic edema and a 0.4 cm midline shift.

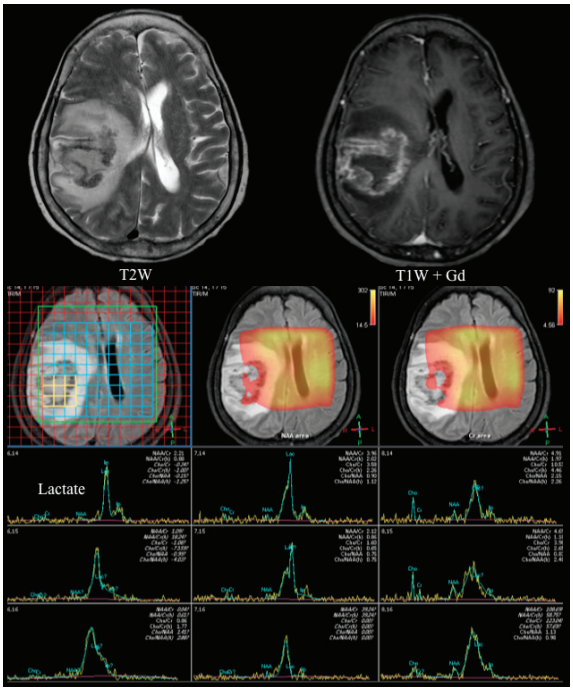

Fig 2–MRI of the brain of the first patient after 1 week of ceftriaxone and clindamycin (T2W, T1W with gadolinium) showing a high SI on T2W, iso-SI on T1W with peripheral rim enhancement in the right frontotemporoparietal region and an increase in the degree of midline shift with right uncal herniation (A). Presence of lactate peak with decreased choline, creatine, and NAA on MRS (B).

cancer. She had underlying hypertension, chronic atrial fibrillation, a history of a right internal capsule cerebral infarction and a history of rheumatoid arthritis she was taking warfarin, simvastatin, atenolol and chloroquine. On examination, her temperature was 39.2ºC and her respiratory rate was 22/minute. The patient was lethargic but able to follow commands. Neurological examination revealed gaze preference to the right, spastic tone and hyperreflexia on her left side with motor strength of 3/5 on the left and 5/5 on the right. Plantar reflexes exhibited an extensor response on the left. No stiffness of the neck was detected. A complete blood count showed a hemoglobin of 10.4 g/dl with a white blood cell count of 10,910 cells/mm^3 (neutrophils 82%, lymphocytes 7%, monocytes 11%). The prothrombin time showed on initial normalized ratio (INR) of 1.8. Computed tomography (CT) of the brain after administration of contrast material showed a hypodense lesion without abnormal enhancement of the right frontotemporoparietal lobe with surrounding vasogenic brain edema and midline shift (Fig 1). The brain CT suggested an acute right middle cerebral artery infarction. A chest radiograph showed an alveolar infiltration of the right lower lung field; ceftriaxone and clindamycin were started to treat aspiration pneumonia.

Blood cultures taken before initiation of antimicrobial therapy grew out a gram-positive bacterium with a positive motility test at ambient temperature, subsequently

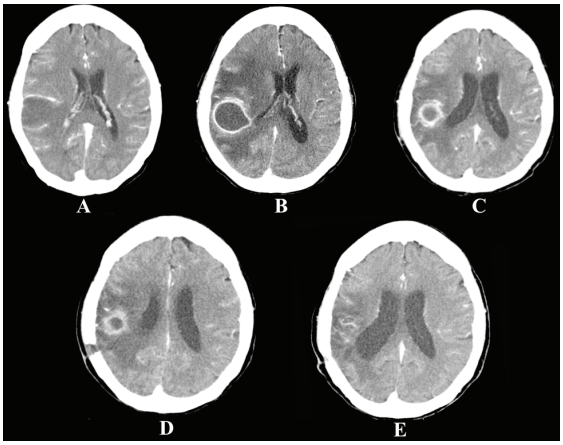

Fig 3–Sequential CT brain images with contrast in the first patient showing a reduction in the abscess size, and a disappearance of rim enhancement after 6 months of antimicrobial therapy. A) At diagnosis; B) at 1 month; C) at 2.5 months; D) at 3.5 months; E) at 6 months.

identified as *L. monocytogenes*. The patient was treated with intravenous ampicillin (12 grams per day). Magnetic resonance imaging (MRI), angiography (MRA) and spectroscopy (MRS) demonstrated cerebritis and an early brain abscess in the right frontotemporoparietal carebrum with right uncal herniation. The lesion was characterized by isosignal intensity (SI) on T1, hypersignal SI on T2 and FLAIR; restrictive diffusion on DWI, peripheral rim enhancement after gadolinium administration, and the presence of a lactate peak, a decreased choline and creatine and a N-Acetyl Aspartate (NAA) peak on MRS (Fig 2). Stereotactic-guided aspiration of the brain was done, due to an increased size in the abscess after five weeks of treatment. Eight milliliters of pus was obtained from the abscess and a Gram's stain of the pus showed numerous polymorphonuclear cells with no organisms. Aerobic and anaerobic cultures yielded no growth. However, molecular identification of the pus using polymerase chain reaction (PCR) and sequencing identified the DNA of *Listeria* spp. The patients was treated with intravenous ampicillin for 10 weeks and switched to oral amoxicillin (3 grams per day) for 6 months. Her weakness abated within 2 weeks and she could walk without support. A CT of the brain after 6-months of treatment showed nearly complete resolution of the lesion, with residual contrast enhancement of the right parietotemporal region (Fig 3).

Case 2

A 47-year-old female with Evan's syndrome (autoimmune hemolytic anemia with idiopathic thrombocytopenic purpura), autoimmune hepatitis, and diabetes mellitus presented with headache, a complex partial seizure and painful subcutaneous nodules of 2 weeks duration. She was being treated with prednisolone at 30 mg/day. On physical examination, she was afebrile with painful erythematous subcutaneous nodules of both her cheeks and the submandibular area. Neurological examination revealed impaired attention but was otherwise normal. A complete blood count revealed a hemoglobin of 9.1 g/dl with a white blood cell count of 2,670 cells/mm^3 (neutrophils 87%, lymphocytes 8%, monocytes 5%), and a platelet count of 52,000 cells/mm^3. A CT of the brain showed effacement of the sulci and gyri in the cortical part of posterior left frontal region and the anterior right temporal region, with gyral enhancement suggestive of cerebritis (Fig 4). Lumbar puncture was performed and the cerebrospinal fluid (CSF) was normal. CSF cultures for bacteria, fungi, and mycobacteria were negative. MRI and MRS of the brain revealed conglomerated lesions with satellite rim enhancing lesions in the cortical and subcortical regions of left middle frontal gyrus and right superior temporal gyrus. Leptomenigeal

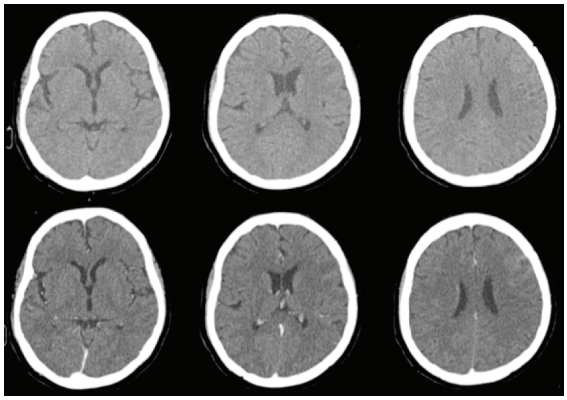

Fig 4–CT brain with contrast of the second patient showing effacement of the sulci and gyri of the cortical part of the posterior left frontal region and the anterior right temporal region, and gyral enhancement after contrast medium injection. All of these findings were suggestive of cerebritis.

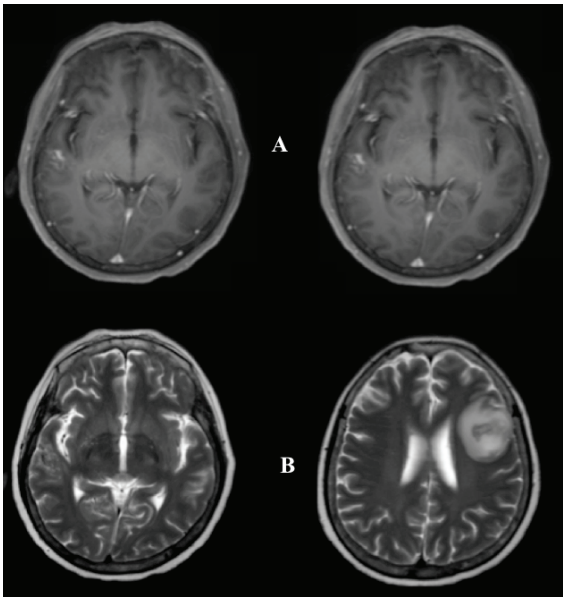

Fig 5–MRI brain of the second patient showing rim-enhancing conglomerated lesions in the cortical-subcortical left middle temporal gyrus and right superior temporal gyrus. These lesions appear as a completely dark rim on T2WI showing a uniform enhancing rim after gadolinium administration. Perilesional vasogenic edema is seen. A) T1WI with gadolinium; B) T2WI.

enhancement of the left frontal lobe was also present (Fig 5). A marked elevation in the lipid/lactate peak with normal choline and the NAA peak on MRS was demonstrated. Those findings were more compatible with brain abscess rather than brain tumor. After the MRI result a hemoculture was obtained and *L. monocytogenes* was recovered. A subcutaneous nodule was biopsied and revealed neutrophilic panniculitis with negative stains, cultures, and PCR results for microorganisms. The patient was treated with ampicillin 12 grams/day IV and phenytoin. Because of the risk of bleeding, a stereotactic brain biopsy was not done. The patient had no further seizures and the erythematous nodules subsided entirely. A follow-up MRI 4 weeks after treatment showed significant improvement in the abscess at the left frontal lobe. Ampicillin was continued for 6 weeks and then switched to oral amoxicillin at 4.5 grams daily.

DISCUSSION

L. monocytogenes is a facultative anaerobic gram-positive non-spore forming rod that is widely distributed in nature and found in multiple ecological sites. *Listeria* is an intracellular organism that can invade tissues normally resistant to infection, such as the central nervous system (CNS), a gravid uterus or a fetus (Gellin and Broome, 1989). Protection against *L. monocytogenes* infection is predominantly cell-mediated (Mielke *et al*, 1997). Therefore, conditions associated with impaired cellular immunity, such as lymphoma, AIDS and corticosteroid

Table 1
Summary of published reports of patients with listerial brain abscesses.

Cases	Age/Sex	Underlying diseases	Immuno-suppressive therapy	Blood C/S	CSF C/S	Surgery	Antibiotics used	Outcomes	References
1	70/M	Alcoholism	None	+	+	ND	ABPC, GM, VCM	Died	(Cone *et al*, 2003)
2	56/M	AIDS	None	+	-	ND	ABPC, GM	Survived	(Patey *et al*, 1989)
3	49/M	Rheumatic fever, alcoholism, diabetes mellitus	None	+	-	ND	PGS	Died	(Buchner and Schneierson, 1968)
4	64/M	DM, aortic valve replacement	None	+	-	ND	ABPC, GM	Survived	(Soto and Sliman, 1992)
5	71/M	Diabetes mellitus, rheumatic heart disease, endocarditis	None	+	-	ND	ABPC, GM	Died	(Eckburg *et al*, 2001)
6	56/M	AIDS	None	+	-	ND	ABPC, GM	Survived	(Patey *et al*, 1989)
7	70/F	Cirrhosis, diabetes mellitus, heart failure	None	+	ND	ND	ABPC, ST	Died	(Sivalingam *et al*, 1992)
8	25/F	Ulcerative colitis	None	NR	NR	ND	NR	Died	(Larsson and Linell, 1979)
9	87/M	None	None	+	+	ND	PGS, CP	Died	(Spilkin *et al*, 1968)
10	63/M	None	None	+	-	ND	ABPC	Died	(Kennard *et al*, 1979)
11	24/M	None	None	+	-	ND	ABPC, GM	Survived	(Smiatacz *et al*, 2006)
12	53/F	None	None	+	-	ND	Minocycline, GM	Survived	(Mrowka *et al*, 2002)
13	63/F	None	None	-	-	ND	NR	Died	(Brun-Buisson *et al*, 1985)
14	43/F	None	None	-	-	ND	ABPC	Died	(Brun-Buisson *et al*, 1985)
15	39/M	None	None	NR	-	ND	NR	Died	(Kwantes and Isaac, 1971)
16	54/F	None	None	NR	NR	ND	NR	Died	(Larsson and Linell, 1979)
17	1 1/4/M	None	None	NR	NR	NR	Amoxicillin	Survived	(Mancini *et al*, 1990)
18	70/M	None	None	-	-	+	ABPC	Survived	(Salgado *et al*, 1996)
19	53/M	Cirrhosis, seizure	None	+	-	+	PGS, EM	Survived	(Halkin *et al*, 1971)
20	85/M	Diabetes mellitus, gout	None	+	-	+	ABPC	Died	(Brown *et al*, 1991)
21	43/M	Sleep apnea syndrome, alcohol abuse	None	+	-	+	NR	Survived	(Douen and Bourque, 1997)
22	0/M	Pronatis	None	+	-	+	ABPC, GM	Survived	(Banerji and Noya, 1999)
23	63/M	Multiple myeloma	NR	+	+	+	ABPC, linezolid	Survived	(Leiti *et al*, 2005)
24	61/M	Diabetes mellitus	None	NR	NR	+	ST, CP	Survived	(Sjostrom *et al*, 1995)
25	60/M	HIV	None	NR	NR	+	PGS, CP	Died	(Harris *et al*, 1989)
26	68/M	Leukemia	NR	NR	NR	ND	CP	Died	(Larsson *et al*, 1978)
27	NR/M	None	NR	NR	NR	ND	NR	Died	(Pollock *et al*, 1984)
28	2/M	NR	NR	NR	+	+	NR	Survived	(Umenai *et al*, 1978)
29	49/M	Renal transplant	AZP, PSL	+	+	ND	CP	Died	(Crocker and Leicester, 1976)
30	16/M	ALL	CMT	+	+	ND	PGS, CP	Survived	(Dykes *et al*, 1979)

31	20/M	ALL	CMT	+	+	ND	ABPC, CP, EM, GM	Survived	(Hutchinson and Heyn, 1983)
32	6/F	ALL	CMT	+	+	ND	ABPC, VCM, netilmicin	Survived	(Viscoli *et al*, 1991)
33	40/F	Ulcerative colitis	PSL	+	+	ND	ABPC, GM	Survived	(Soares-Fernandes *et al*, 2008)
34	58/F	Multiple endocrine neoplasm 2A, SLE	PSL	+	-	ND	PGS, TOB	Survived	(Takano *et al*, 1999)
35	58/F	Immunoblastic lymphadenopathy	PSL, CMT	+	-	ND	ABPC	Survived	(Maezawa *et al*, 2002)
36	65/M	Diabetes mellitus	PSL	+	NR	ND	ABPC, GM	Survived	(Wu *et al*, 2010)
37	19/M	Juvenile rheumatoid arthritis, Tetralogy of Fallot	PSL	NR	+	ND	VCM, ABPC	Survived	(Turner *et al*, 1995)
38	55/M	Renal transplant	AZP, PSL	+	-	+	ABPC	Survived	(Lechtenberg *et al*, 1979)
39	45/M	Renal transplant	AZP, PSL	+	-	+	ABPC	Survived	(Stam *et al*, 1982)
40	60/F	Rheumatoid arthritis	PSL	+	-	+	ABPC	Survived	(Updike *et al*, 1990)
41	66/F	AML, Crohn's disease	PSL	+	-	+	ABPC, ST	Survived	(Eckburg *et al*, 2001)
42	47/M	AIDS, Multiple liver abscess, *M.avium* bacteremia, CMV retinitis	Ganciclovir	+	-	+	ABPC, GM, VCM	Died	(Cone *et al*, 2003)
43	54/F	Sarcoidosis	PSL	+	-	+	ABPC, GM	Died	(Ackermann *et al*, 2001)
44	23/F	ITP	PSL, CY	+	-	+	ST	Survived	(Treebupachatsakul *et al*, 2006)
45	58/M	Multiple myeloma	VAD	+	-	+	ABPC, GM	Survived	(Al-Khatti and Al-Tawfiq, 2010)
46	55/M	Glioblastoma multiforme	CMT	-	+	+	ABPC, GM	Survived	(Ganiere *et al*, 2006)
47	51/M	Cardiac transplant	AZP, cyclosporine, PSL	+	ND	+	ABPC, GM	Survived	(Eckburg *et al*, 2001)
48	37/M	Cardiac transplant	PSL, cyclosporine	+	ND	+	ABPC	Survived	(Eckburg *et al*, 2001)
49	56/F	Autoimmune hepatitis, primary biliary cirrhosis	AZP, PSL	+	ND	+	ABPC, GM	Survived	(Cone *et al*, 2003)
50	50/M	Sarcoidosis	PSL	-	-	+	ST	Survived	(Poropatich and Phillips, 1992)
51	51/F	Crohn's disease	PSL	-	-	+	ABPC, GM	Survived	(Stefanovic *et al*, 2010)
52	50/M	Cardiac transplant, diabetes mellitus perirectal abscess	AZP, cyclosporine, PSL	-	-	+	ABPC, GM	Died	(Eckburg *et al*, 2001)
53	75/M	COPD	PSL	-	ND	+	ABPC, GM	Survived	(Mylonakis *et al*, 1998)
54	77/M	CLL	CMT	-	NR	+	CP	NR	(Cleveland and Gelfand, 1993)
55	58/M	CLL	CMT	ND	ND	+	ABPC, GM	Survived	(Dee and Lorber, 1986)
56	Child	ALL	CMT	NR	NR	+	NR	Survived	(Antunes *et al*, 1998)
57 (PR)	58/F	Breast cancer	CMT	+	ND	+	ABPC	Survived	
58 (PR)	47/F	Evan's syndrome, SLE, diabetes mellitus	PSL	+	-	ND	ABPC	Survived	

ABPC, ampicillin; AIDS, acquired immune deficiency syndrome; ALL, acute lymphoblastic leukemia; AML, acute myeloid leukemia; AZP, azathioprine; CLL, chronic lymphocytic leukemia; CMT, chemotherapy; CP, chloramphenicol; CY, cyclophosphamide; EM, erythromycin; GM, gentamicin; ITP, immune thrombocytopenic purpura; ND, not done; NR, not reported; PGS, penicillin G sodium; PR, present report; PSL, prednisolone; SLE, systemic lupus erythematosus; ST, trimethoprime/sulfamethoxazole; TOB, tobramycin; VAD, vincristine doxorubicin and dexamethasone; VCM, vancomycin

use are risk factors for listerial infection (Stam *et al*, 1982; Southwick and Purich, 1996). Ingestion of *L. monocytogenes* contaminated food is considered to be the source of nearly all listerial human infections (Lorber, 1997). Once ingested, *L. monocytogenes* penetrates the Peyer's patches of the small intestine, but not via the phagocytic microfold or M cells (Pron *et al*, 1998). The bacterium enters the mesenteric lymph nodes and then the blood stream, resulting in bacteremia (Cone *et al*, 2003). Meningitis may occur when the organisms attach to the epithelial cells of the choroid plexus (Schluter *et al*, 1996). Cerebritis and subsequent brain abscesses result from penetration of the bacterium into the brain parenchyma through the cerebral capillary endothelium (Kirk, 1993; Dramsi *et al*, 1998); the infected macrophages penetrate these endothelial cells via the cerebral artery (Muller and Weigl, 1992). *Listeria* displays a tropism for the CNS, with manifestations ranging from meningitis to cerebritis and focal parenchymal involvement (Cone *et al*, 2003). A unique form of listerial parenchymal CNS infection is rhombencephalitis, an acute brainstem infection characterized by asymmetrical cranial nerve palsies, cerebellar dysfunction, hemiparesis, impairment of consciousness and possible respiratory failure (Armstrong and Fung, 1993). Listerial abscesses of the cerebral hemisphere are extremely rare, accounting for approximately 1-10% of listerial CNS infections (Nieman and Lorber, 1980; Lorber, 1997; Cone *et al*, 2003). Listerial brain abscesses are associated with meningitis in up to 38% of the patients (Chun *et al*, 1986). Involvement of the subcortical grey matter, such as the thalamus and basal ganglia, is more common in listerial brain abscesses (about 21%) than with other agents (Lorber, 1997). Similar to other bacterial brain abscesses, most patients with listerial brain abscesses have fever, elevated leukocyte counts, headache, and focal neurological signs. Bacteremia is common and found in about 86% of *Listeria* brain abscess (Dee and Lorber, 1986). Bacteremia is unusual in brain abscesses caused by other organisms, in which it occurs in approximately 11% of cases (Mathisen and Johnson, 1997). Hence, three different features of listerial brain abscesses are: 1) the presence of bacteremia in most patients, 2) a quarter of patients exhibit concomitant meningitis, and 3) subcortical abscesses are usually located in the pons, thalamus, or medulla (Lorber, 1997). However, listerial brain abscesses may mimic other causes of CNS disease, such as strokes or brain tumors; an MRS of the brain may be useful test to distinguish the etiologies (Lai *et al*, 2002). Choline is a marker of increased cellular turnover and is elevated in tumors and gliosis, but is decreased in abscesses (Gujar *et al*, 2005). Lactate, a product of anaerobic glycolysis, is detected in any diseased brain, such as with an infarction, tumor or infection (Gujar *et al*, 2005). NAA, a neuronal marker, and creatine, a measure of energy stores, are decreased in cases of infarctions, tumors, and infections (Gujar *et al*, 2005).

There have been no controlled trials to establish a drug of choice or duration of therapy for listerial infection (Lorber, 1997). Ampicillin is generally considered as the preferred agent for treatment of listeriosis, although its superiority to penicillin is questionable. For patients intolerant of penicillins, trimethoprim-sulfamethoxazole is thought to be the best alternative single agent (Winslow and Pankey, 1982; Treebupachatsakul *et al*, 2006). On the basis of the synergy observed with *in vitro* and in animal models (Edmiston

and Gordon, 1979), most authorities suggest adding gentamicin to ampicillin to treat listerial bacteremia in patients with severely impaired T-cell function and in all cases of listerial meningitis and endocarditis (Nieman and Lorber, 1980; Gellin and Broome, 1989; Cherubin *et al*, 1991). Linezolid and rifampicin are active against *L. monocytogenes in vitro* and cross the blood-brain barrier. Combination treatment involving rifampin and another active antimicrobial may reduce the emergence of resistance to rifampin (Leiti *et al*, 2005). Leiti *et al* (2005) reported successful treatment with a combination of linezolid and rifampin in the case of a listerial brain abscess without any adverse events. Patients with a brain abscess require treatment for at least six weeks and should be followed up with serial neurological imaging until the abscesses are resolved (Lorber, 1997). The mortality rate in listerial CNS abscesses has been reported to be 40% compared with 17% for other types of brain abscesses (Cone *et al*, 2003).

To our knowledge, only 56 cases of macroscopic brain abscesses due to *L. monocytogenes* have been reported between 1968 and 2011 (Table 1). Thirty-nine patients (68%) were men. The mean age was 50±20 years (range 0-87 years). Most patients (80%) had underlying conditions, such as a hematological malignancy (23%), autoimmune diseases treated with prednisolone (19%), having undergone solid organ transplant (10%), diabetes mellitus (12%), human immunodeficiency virus (HIV) infection (7%), or others (9%). Two of our patients had an underlying malignancy and an autoimmune disease treated with corticosteroids and immunosuppressive agents, respectively. Positive blood cultures and CSF cultures were found in 79% and 23%, of cases listed in Table 1, respectively. Ampicillin-based regimens were used in 37 patients (74%). Twenty-nine patients (51%) underwent surgery. A total of 19 patients died, giving a mortality rate of 33%. Patients who had underlying diseases and who did not undergo surgical interventions were more likely to die (63% and 74%, respectively).

In conclusion, due to the severity of listerial diseases, its predilection for the CNS and high mortality rate, clinicians should retain a high index of suspicion in susceptible patients presenting with CNS infections. Blood cultures are usually positive in patients with listerial brain abscesses. A MRS of the brain may be beneficial to differentiate between a brain tumor and an abscess. Ampicillin should be considered as part of combination antimicrobial therapy in such patients.

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