Clinical Presentation and Cranial MRI Findings of *Listeria monocytogenes* Encephalitis

A Literature Review of Case Series

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Background: *Listeria monocytogenes*–associated encephalitis is a severe clinical condition that can also be seen in immunocompetent patients. Clinical manifestation and radiologic features of this entity need to be elaborated.

Review Summary: We searched the medical literature during the period spanning from 1991 to 2017 using the keyword "listeria AND (abscess OR brainstem OR encephalit* OR magnetic* OR imaging*)." We included in the review well-documented adult cases with a definitive diagnosis and having magnetic resonance imaging data. Confusion, hemiparesis, cerebellar ataxia, facial paralysis, and gait disturbance were the most frequent findings, detected in >30% of patients during admission. The high rate of facial paralysis was of particular interest. T2 hyperintensity (80/82), contrast enhancement (60/82), and ring-enhancing lesions, which are considered as brain abscess, were found in 46/82 patients. The mortality and neurological sequel rates were 20% and 68%, respectively.

Conclusions: Listeria encephalitis is a severe disease and should be remembered in cases admitted with symptoms related to the brainstem and cranial nerve dysfunction. Cranial magnetic resonance imaging

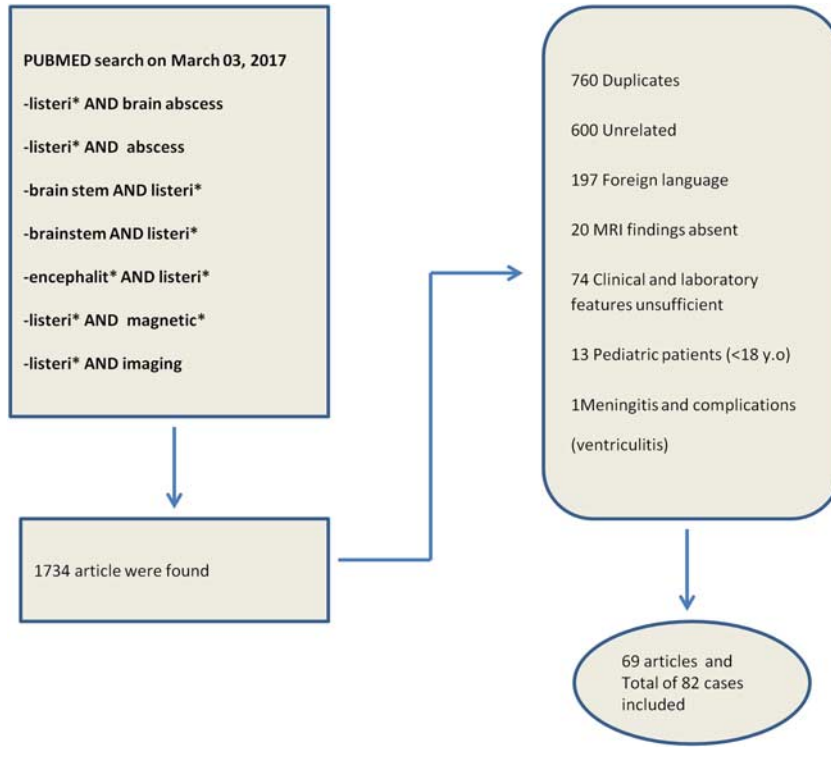

FIGURE 1. Flowchart selection process of case reports, exclusion criteria, and included cases.

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with brainstem and cerebellum involvements and contrast enhancement with or without abscess are particularly suggestive of the listeria-related infection.

Key Words: brain stem, encephalitis, listeria, magnetic resonance imaging, meningitis, rhombencephalitis

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Listeria monocytogenes (LM) is a facultative anaerobic, intracellular bacillus gram-positive.[1] The gastrointestinal system is the main route of LM infection. The most common clinical form is self-limited gastroenteritis.[2] LM also shows central nervous system (CNS) tropism. CNS infections of LM consist of meningitis, encephalitis, and brain abscess.[3–5] The terms rhombencephalitis or brainstem encephalitis is referred if LM infection is restricted to the medulla oblongata, mesencephalon, pons, or cerebellum.[6,7]

As LM CNS infections are rare, data on imaging findings and clinical presentation are scarce. However, the developments in imaging techniques, culture, and molecular detection methods during the last few decades have the potential to increase our understanding and awareness of the diagnosis of LM CNS infections.

We reviewed LM encephalitis cases from the medical literature and analyzed reports having a definitive diagnosis and magnetic resonance imaging (MRI) data in the English medical literature during the period spanning from 1991 to 2017.[8–75] We evaluated demographics, clinical presentations, and MRI features of cases. The 11 cases of these patients were retrieved from our previously puplished manuscript.

MATERIALS AND METHODS

Literature Search Strategy

We searched the medical literature during the period spanning from 1991 to 2017 using the keywords "listeria AND [abscess odds ratio (OR) brainstem OR encephalit* OR magnetic* OR imaging*]." Figure 1 illustrates the data-searching and selection strategy. We included only well-documented cases that had a definitive diagnosis based on positive cultures, complement fixation test (1 patient), or positive polymerase chain reaction results. We included only the data of adults (older than 18 years old) with MRI findings. Ethical approval was not applicable.

Clinical and Laboratory Features

We evaluated patients having a severe underlying disease such as a malign disease, transplantation, chronic renal failure, diabetes mellitus, alcoholism, cirrhosis, or HIV infection or those who were using certain drugs such as corticosteroids or monoclonal antibodies.

We extracted clinical signs and symptoms from the reports. Fever plus nuchal rigidity plus a headache were recorded as "classical triad." Cerebrospinal fluid (CSF) analyses (cell count, glucose, protein) were recorded if available.

Neurological findings and sequelae were extracted and categorized into groups: (i) motor nerves, (ii) cranial nerves, (iii) cerebellar dysfunction, and (iv) others (aphasia, convulsion, arrhythmia).

We defined death or persistent neurological sequelae as an unfavorable outcome.

Cranial MRI Assessment

Data on conventional MRI signal pattern (T2 hyperintensity), mass effect, expansion, edema, abscess formation, hemorrhage, and hydrocephalus were extracted from studies. CNS lesions were recorded according to the involved sites, as well. An experienced neuroradiologist (PhD) scrutinized the data.

Statistical Analyses

Statistical analyses were accomplished with the open source statistical package R. Continuous variables were presented as means and SDs or medians and interquartile ranges (IQR) according to the normal or non-normal distributions, respectively. Categorical variables were presented as frequencies and percentages and, where available, χ^2, test or the Fisher exact test were used for comparisons.

RESULTS

Demographic, Clinical, and Laboratory Features

Data of 82 patients were pooled from 70 discrete reports. The mean age was 54 ± 15 years, and the frequency of male sex was 56% (46/82). Immunocompromised conditions were detected in 52% (43/82) of patients, and long-term corticosteroid intake was the most prevalent cause (67%, 29/43). Cirrhosis (n = 9) and inflammatory bowel diseases (n = 6) were noticeable underlying diseases. Classical triad of meningitis (fever, headache, and nuchal rigidity) was found in only 15% of the patients.

Laboratory results of various components of CSF were reported for 66 patients. The overall CSF analysis were as follows: cell count (median, 101; IQR, 40 to 309; n = 61),

TABLE 1. Neurological Symptoms and Findings of *Listeria monocytogenes* Encephalitis

	n (%)		
Neurological Findings/Sequelae	**At Admission**	**Healed***	**Developed (N)†**
Confusion	35 (43)	22 (63)	—
Hemiparesia	33 (40)	16 (48)	2
Cerebellar ataxia	32 (39)	23 (72)	—
Facial paralysis	30 (37)	20 (67)	2
Gait disturbance	26 (32)	20 (77)	2
Nystagmus	23 (28)	21 (91)	—
Respiratory failure	23 (28)	0 (0)	—
Coma	21 (26)	10 (48)	—
Difficulty in swallowing	18 (22)	14 (78)	2
Diplopia	17 (21)	14 (82)	2
Dysphagia	17 (21)	13 (76)	2
Abducens paralysis	15 (18)	10 (67)	1
Dysarthria	14 (17)	9 (64)	—
Convulsion	9 (10)	5 (56)	1
Trigeminal paralysis	9 (10)	7 (78)	—
Dysmetria	8 (9)	7 (87)	2
Vertigo	8 (9)	8 (100)	—
Aphasia	7 (8)	6 (86)	—
Glossopharyngeal paralysis	7 (9)	5 (71)	—
Anisocoria	5 (6)	4 (80)	—
Tetraplegia	5 (6)	5 (100)	2
Vagus paralysis	5 (6)	5 (100)	—
Oculomotor paralysis	4 (5)	3 (75)	2
Hyoglossus paralysis	2 (2)	1 (50)	—
Trochlear paralysis	2 (2)	2 (100)	—
Accessorius paralysis	1 (1)	1 (100)	—
Arrhythmia	1 (1)	—	—
Vestibulocochlear paralysis	1 (1)	—	—

*The healed percentage is referring to the presented frequencies.
†Developed among the survivors during the course of the disease.

TABLE 2. Univariate Comparison of Demographic and Clinical Variables Between Outcomes

	Outcome [n (%)]			
	Favorable (N = 36)	**Unfavorable (N = 46)**	**OR**	***P***
Age (y)	55.2 (12.3)	52.8 (17.2)	0.99 (0.96-1.02]	0.456
Male sex	17 (63)	29 (52.7)	1.51 (0.59-4.02)	0.552
Sypmtoms and signs				
Confusion	12 (33.3)	23 (51.1)	2.06 (0.84-5.27)	0.168
Comatose	5 (13.9)	16 (34.8)	3.21 (1.09-11.1)	0.058
Convulsion	2 (5.6)	7 (15.2)	2.87 (0.62-22.4)	0.286
Anizokoria	3 (8.3)	2 (4.3)	0.52 (0.06-3.57)	0.650
Diplopia	5 (13.9)	12 (26.1)	2.14 (0.69-7.55)	0.281
Nystagmus	8 (22.2)	15 (32.6)	1.67 (0.62-4.78)	0.429
Vertigo	2 (5.6)	6 (13.0)	2.41 (0.50-19.2)	0.456
Cerebellar ataxia	12 (33.3)	20 (43.5)	1.53 (0.62-3.88)	0.480
Gait disturbance	10 (27.8)	16 (34.8)	1.38 (0.53-3.68)	0.662
Facial hypoestesia	6 (16.7)	12 (26.1)	1.73 (0.59-5.63)	0.451
Disphagia	7 (19.4)	10 (21.7)	1.14 (0.38-3.57)	1.000
Difficulty with swallowing	8 (22.2)	10 (21.7)	0.97 (0.33-2.89)	1.000
Aphasia	4 (11.1)	3 (6.5)	0.57 (0.10-2.90)	0.694
Disarthria	5 (13.9)	9 (19.6)	1.48 (0.45-5.41)	0.702
Dysmetria	4 (11.1)	4 (8.7)	0.76 (0.16-3.63)	0.725
Cranial nerve palsy				
Oculomotor	2 (5.6)	2 (4.3)	0.78 (0.08-7.76)	1.000
Trochlear	1 (2.8)	1 (2.2)	0.78 (0.02-31.2)	1.000
Trigeminal	2 (5.6)	7 (15.2)	2.87 (0.62-22.4)	0.286
Abducens	2 (5.6)	13 (28.3)	6.21 (1.53-45.8)	0.019
Facial	12 (33.3)	18 (39.1)	1.28 (0.51-3.26)	0.757
Glossopharyngeus	2 (5.6)	5 (10.9)	1.98 (0.38-16.1)	0.458
Vagus	2 (5.6)	3 (6.5)	1.16 (0.17-10.4)	1.000
Motor nerve palsy				
Hemiparesia	12 (33.3)	21 (45.7)	1.66 (0.67-4.23)	0.367
Tetraplegia	2 (5.6)	3 (6.5)	1.16 (0.17-10.4)	1.000

OR indicates odds ratio.

lymphocytic pleocytosis (69%, n = 49), glucose level (mean, 55 ± 30 mg/dL, n = 40), and protein level (median, 100 mg/dL; IQR, 60 to 185 mg/dL; n = 55). LM was cultured from 27 (41%) and 60 (79%) of CSFs and blood cultures, respectively.

Totally 17 (20%) patients died. Of the 65 patients who survived, 58% (n = 38) had neurological sequelae. Table 1 presents neurological symptoms and findings at admission, during the course, and at discharge. Confusion, hemiparesis, cerebellar ataxia, facial paralysis, and gait disturbance were the most prominent findings, detected in > 30% during admission. It was noteworthy that facial paralysis was observed at a high rate.

Table 2 presents univariate comparisons of demographic and clinical variables between favorable and unfavorable outcomes. All the variables were equal between outcomes except being in a coma state and having VI cranial nerve (nervus abducens) palsy at admission. These were observed more commonly among patients with unfavorable outcomes.

Radiologic Features

A total of 139 distinct lesions had been reported. Table 3 presents cranial MRI features. Briefly, T2 hyperintensity (81/82), contrast enhancement (60/82), and ring-enhancing lesions, which were considered as brain abscess (46/82), were common in patients. Mass effect lesions were reported in 8 (10%) patients; 3 of them were located in the brainstem, 3 in the cerebellar hemisphere, 1 in the cerebellar peduncle, 1 in the ventricular basement, and 1 in the frontal lobe. Two of them were located in the frontal and parietal white matter. Nearly half of the mass effect lesions has contrast enhancement, and some were extending from white matter to corpus callosum. Edema

TABLE 3. Cranial MRI Feautures of *Listeria monocytogenes* Encephalitis Cases

MRI Findings	n (%)	References
T_2 hyperintensity	81	8–61,63–75,77
Contrast enhancement	60	8–18,23,24,27,28,30–34,36,37,39,40,42,43,45,46,48,49,51,52,55–60,63–68,70,71,73–75,77
Abscess	46	8,11–17,24,30,32–34,36,37,39,40,42,43,45,46,48,49,51,52,54–56,59,63,65–67,70,71,73–75,77
Edema	16	8,13,27,35,39,46,52,54,57,64,66,68,69,73–75
Mass effect	8	8,18,21,38,49,57,69,74
Hydrocephalus	7	8,10,17,29,35,43,77
Expansion	4	18,21,27,61
Hemorrhage	4	30,64,66,73

MRI indicates magnetic resonance imaging.

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TABLE 4. Localization of Lesions in Cranial Magnetic Resonance İmaging

Region of Lesions	n	References
Brain stem	55	11–24,27–33,35–37,39–45,47–51,53,56,59,62,66,68,70,75,77
Supratentorial white matter	25	9,22,34,35,40,45,46,48,54,57,58,60,61,63–66,68,69,71–74,77
Cerebellar hemisphere	20	8,12,17,18,20,21,26,27,37,38,43,50,52,59,70,73,77
Cerebellar peduncle	13	10–12,15,18,25,26,36,37,41,44,68
Basal ganglia	10	9,13,26,35,37,45,53,55,67,77
Internal capsule	7	26,42,49,53,55,68,69
Thalami	4	13,24,45,75
Spinal cord	4	10,15,37,51
Supratentorial cortex	1	52
Periaqueductal gray matter	1	19
Vermis	1	20
Trigeminal nerve	1	28

was detected in 2 of the mass effect lesions regardless of contrast enhancement.

Table 4 presents the localization of lesions in cranial MRI. Brainstem (55/82), supratentorial white matter (25/82), cerebellar hemisphere (20/82), and cerebellar peduncle (13/82) were the most frequently involved regions. Two brainstem lesions were hemorrhagic, and hematoma was detected in the cerebellum.

Treatment Features

Patients mostly have been treated with ampicillin as monotherapy (17/82) or a beta-lactam plus aminoglycoside combination (48/82). In univariate analysis, neither ampicillin monotherapy [OR, Confidence interval (CIs); 0.34 (0.11-1.02)] nor combination therapy [OR, CIs; 1.20 (0.47-3.05)] were statistically significant between outcomes. Long-term corticosteroid use was not a significant predisposing factor for unfavorable outcomes [OR, CIs; 0.34 (0.11-1.02)].

DISCUSSION

This review documented that confusion, hemiparesis, cerebellar ataxia, facial nerve palsy, and gait disturbance were the most common signs detected at the admission of patients with LM CNS infection. Detection of facial nerve palsy at admission is particularly noteworthy, as, to our knowledge, the English literature does not mention facial nerve paralysis as a common sign of LM encephalitis.[76]

The second noticeable finding in this review was that nearly half of the patients presented with cranial nerve palsy (37/82). However, none of the findings except nerve abducens palsy were related to an unfavorable outcome. Nerve abducens palsy, although 67% of them healed during the disease course, was significantly common among unfavorable outcomes.

This study provided valuable information on MRI findings. Studies reported T2 hyperintensity among almost all patients. Most noteworthy, in our opinion, was the high detection rate of contrast enhancement with or without abscess. As expected, the brainstem was the most commonly involved site, followed by supratentorial white matter and cerebellar hemisphere.

The mortality rate in our study (20%) was comparable with other studies (13% to 29%).[77] However, neurological sequelae rate was very high (68%) and might frequently relate to parenchymal involvement.

MRI has clear advantages to CT in detecting the listerial CNS infections. LM should be remembered in cases wherein brainstem involvement is detected on cranial MRI. Tuberculosis, aspergillosis, paracoccidioidomycosis, Human herpesvirus 6, enterovirus 71, Behcet disease and systemic lupus erythematosus might cause brainstem involvement, as well.[78]

Treatment regimens of LM encephalitis do not differ from treatment regimens of LM meningitis, as cerebral drug bioavailability of antibiotics is usually similar.[79] In this review, we found that ampicillin monotherapy and aminoglycoside combination seem to have similar outcome numbers.

In conclusion, listeria encephalitis is a severe disease and should be remembered in cases of patients admitted with signs of brainstem dysfunction and cranial nerve dysfunction. Cranial MRI with brainstem and cerebellum involvements with contrast enhancement or abscess is particularly suggestive of LM CNS infections.

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