



ORIGINAL ARTICLE

Encephalitis and Meningoencephalitis in Children in Batna, Algeria

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ABSTRACT

Acute encephalitis in children most often occurs in the context of a systemic infection; however, in some cases, neurological involvement may be the sole manifestation. Infectious encephalitis is more frequent in young children and may be associated with meningeal involvement (meningoencephalitis) or spinal cord involvement (encephalomyelitis). Herpes simplex virus (HSV) is the most common cause of encephalitis in children older than six months, and polymerase chain reaction (PCR) constitutes the cornerstone of diagnosis. **Objectives:** To describe the specific features and frequency of encephalitis and meningoencephalitis in children. **Methods:** From April 1, 2012 to August 31, 2015, a prospective, longitudinal, and exhaustive study was conducted, including patients older than 28 days presenting with clinical features suggestive of encephalitis and meeting the inclusion and exclusion criteria defined by the French Infectious Diseases Society (SPILF). Patients were recruited from three hospital departments (intensive care, infectious diseases, and pediatrics). HIV-positive patients were excluded. **Results:** Among 141 cases, 38 were children (27%). The median age was 5 years (range: 1–9), with a male predominance (22/38; M/F ratio: 1.37). Comorbidities were present in 4 cases (10.5%). Onset was abrupt in 81.6% of cases. Severe presentations were observed, with a Glasgow Coma Scale score below 8 in 18.4% of patients. Seizures and status epilepticus occurred in 73.7% and 36.8% of cases, respectively. Focal neurological deficits were observed in 57.8% and behavioral disturbances in 34.2%. Respiratory involvement was noted in 36.8%. Cerebrospinal fluid pleocytosis was found in 83.8%, elevated protein levels in 51.4%, and hypoglycorrhachia in 32.4%. Neuroimaging was abnormal in 60.5% of cases, and electroencephalography was abnormal in 23.7%. An etiological diagnosis was established in 28 of 38 children (73.7%), including 14 monomicrobial infections (36.8%) and 14 coinfections (36.8%). Viral etiologies predominated: 34 children (89.5%) had at least one confirmed or probable viral cause (coronavirus, EBV, adenovirus, CMV, influenza virus, VZV, enterovirus, HSV, rubella virus, rhinovirus, HHV-6, and parvovirus B19). Nine children (23.7%) had at least one confirmed or probable bacterial etiology. Mortality was 23.7%. **Conclusion:** This study highlights the etiological spectrum of encephalitis and meningoencephalitis in children, along with their clinical and paraclinical characteristics, in eastern Algeria (Batna).

Keywords: post-infectious encephalitis, primary encephalitis, PCR.

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1. INTRODUCTION

Encephalitis refers to inflammatory disorders of the brain parenchyma responsible for neurological dysfunction. These conditions may be of infectious or non-infectious origin and may evolve acutely, subacutely, or chronically (1,2). Infectious encephalitis is more

frequent in young children (3,4) and may be associated with meningeal involvement (meningoencephalitis) (5) or spinal cord involvement (encephalomyelitis) (6-9). It is a rare but severe clinical syndrome caused by a wide range of infectious agents, most commonly viruses (10).

Clinical manifestations may result either from direct aggression of the brain parenchyma or from indirect invasion of the central nervous system by infectious agents. Two main pathogenetic mechanisms are classically distinguished: primary encephalitis, caused by viral replication within the brain parenchyma(11), and post-infectious encephalitis, also known as acute disseminated encephalomyelitis, which results from immune-mediated mechanisms triggered by infection without direct viral invasion of neural cells (12,13). Measles virus and *Mycoplasma pneumoniae* are among the most frequently implicated pathogens. In many cases, it is not possible to clearly distinguish between primary and post-infectious encephalitis, as some pathogens, such as HSV or *Mycoplasma pneumoniae*, may be involved in both mechanisms, complicating diagnosis and management(11).

In children, acute encephalitis most often occurs during a systemic infection, although neurological involvement may occasionally be isolated. Lesions may predominate in either gray or white matter depending on the responsible pathogen and the underlying pathophysiological mechanism. In addition to viruses, bacteria—particularly intracellular organisms—may cause encephalitis in variable proportions depending on published series(7, 14, 15). More recently, the role of autoimmune antibodies has been demonstrated, with clinical presentations similar to those of infectious encephalitis(16).

The incidence of acute encephalitis varies widely according to geographic region and season, reflecting seasonal viral circulation and the restricted distribution of certain pathogens such as Japanese encephalitis virus, Eastern equine encephalitis virus, or West Nile virus. In routine clinical practice, the etiology remains undetermined in a substantial proportion of cases. However, the development of PCR-based techniques has significantly improved diagnostic yield and now represents the cornerstone of etiological diagnosis. (11)

Diagnosis should be suspected in the setting of acute onset of neurological symptoms associated with fever, including altered consciousness, seizures, and focal neurological deficits. HSV is the most frequent cause of encephalitis in children older than six months(17). Post-infectious encephalitis is more common than primary encephalitis, with a peak incidence around 4–5 years of age. Cerebrospinal fluid may be normal in 3–5% of cases (18, 19). Reported mortality ranges from 4% to 12%, and long-term sequelae occur in 25–35% of patients. Etiology remains the main prognostic factor, although it is undetermined in up to 33–85% of cases(20). Certain clinical forms deserve particular attention, including basal ganglia encephalitis (21), rhombencephalitis, neonatal encephalitis (22), and post-infectious Guillain–Barré syndrome(23).

The objective of this study was to describe the specific features and frequency of encephalitis and meningoencephalitis in children in Batna, Algeria.

2. MATERIALS AND METHODS

A prospective, longitudinal, and exhaustive study was conducted from April 1, 2012 to August 31, 2015. Patients older than 28 days and up to 15 years old presenting with clinical features suggestive of encephalitis and meeting SPILF inclusion and exclusion criteria were enrolled from three hospital departments (intensive care, infectious diseases, and pediatrics). Epidemiological, clinical, biological, radiological, and etiological data were collected. HIV-positive patients were excluded.

Inclusion Criteria

Children hospitalized for encephalitis or meningoencephalitis, presenting altered mental state, seizures, central or peripheral neurologic deficits, or autonomic dysfunctions associated with a fever $\geq 38^{\circ}\text{C}$ and a CSF with at least one of the following anomalies: ≥ 4 cellular elements or ≥ 0.4 g/L of proteins, or children of more than 28 days of age hospitalized for neurologic or radiologic signs of febrile encephalitis with a normal CSF, due to the frequency of post-infectious encephalitis. Exclusion criteria were HIV positivity, cerebral abscess, and non-infectious pathologies of the central nervous system.

Biological samples were collected at admission according to standard care protocols for suspected encephalitis. Additional samples for extended microbiological investigations were collected and immediately frozen at -80°C , including EDTA blood, serum, pharyngeal swabs, and cerebrospinal fluid. Follow-up blood samples were obtained at discharge. PCR/RT-PCR assays, viral cultures, and serological tests were subsequently performed at the Institute of Infectious Agents, Lyon University Hospital. Molecular analyses were carried out according to standardized laboratory procedures, with nucleic acid extraction performed using the NucliSENS EasyMAG system (bioMérieux). A total of 2,671 PCR assays, 404 bacterial cultures and direct examinations, 219 viral cultures, and 380 serological tests were performed on all the cohort (141 adults and children), including 724 PCR tests for children. The Ct threshold for the PCR test that was the lowest indicated a high viral load. For CMV, EBV, and HHV6, which are recurrent viruses, Ct value was compared between blood and CSF.

The number of pathogens investigated per patient ranged from 1 to 31. For 13 patients, when the first diagnostic test was positive, one diagnostic test would be performed. As for the rest, it was necessary to perform all the battery of tests. Therefore, the number of tests performed on patients for whom an etiology had been identified is less than for those in which no pathologic agent was identified. Etiological classification followed SPILF recommendations, categorizing diagnoses as confirmed, probable, or possible: confirmed cases are those with positive CSF microbiological results (PCR, direct exam, culture) or positive CSF counter immunoelectrophoresis, probable cases are those with positive diagnostic results in a biological fluid other than the CSF or a CSF serologic test (seroconversion or high IgM titer); possible cases are patients with high serum IgM titer or symptoms or signs compatible with a certain infection associated with an epidemiological context in whom diagnostic tests with a known low sensitivity were negative; unknown etiology cases are patients in whom results of all tests were negative.

Ethical guidelines, including medical confidentiality and obtaining patients' consent for prescribed therapeutic measures, were strictly adhered to throughout the study. Verbal consent for the lumbar puncture procedure was obtained from the patients or their families.

3. RESULTS

During the study period, 141 HIV-negative patients meeting the inclusion criteria for encephalitis and/or meningoencephalitis were enrolled. Among them, 38 were children aged 28 days to 15 years, representing 27% of the total cohort. The median age was 5 years (range: 28 days–14 years), with a male predominance (22/38; male-to-female ratio 1.37). The most affected age group was children aged 28 days to 5 years (57.9%). Most children lived in areas with high population density, including urban (47.4%) and rural (50%) settings. Relevant medical history was identified in 7 cases, including recent animal bite with vaccination, neonatal meningoencephalitis with ventricular shunt, head trauma, chronic sinusitis, immunosuppressive therapy, and corticosteroid treatment. Comorbidities were present in 4 children (10.5%), including two cases of renal disease (one chronic renal failure on dialysis and one nephrotic syndrome), one congenital heart disease, and one cochlear implant. A seasonal predominance was observed during winter (47.4%).

Clinical onset was abrupt in 81.6% of cases. Severe neurological involvement was frequent, with altered consciousness observed in 97.3% and a Glasgow Coma Scale score <8 in 18.4%. Seizures occurred in 73.7% of children, including status epilepticus in 36.8%. Focal neurological deficits were present in 57.8%, and behavioral disturbances in 34.2%. Respiratory involvement was noted in 36.8% of cases.

Cerebrospinal fluid analysis showed pleocytosis in 83.8% of patients, predominantly lymphocytic (56.8%), followed by neutrophilic (32.4%) and mixed patterns (10.8%). Elevated protein levels were observed in 51.4%, and hypoglycorrhachia in 32.4%. Neuroimaging was abnormal in 60.5% of cases, revealing hydrocephalus (18.4%), acute disseminated encephalomyelitis (5.2%), myelitis (2.6%), frontal lobe involvement (21.1%), temporal lobe involvement (15.8%), and tuberculomas (2.6%). Electroencephalography was performed in 22 children (57.9%) and was abnormal in 40.9%, showing epileptiform activity in 22.7% and diffuse slowing in 18.2%. Table 1 summarizes the epidemiological, clinical, biological, and etiological aspects of encephalitis/meningoencephalitis, as well as the outcome of children included in our study.

An etiological diagnosis was established in 28 of the 38 children (73.7%). Fourteen cases (36.8%) were monomicrobial infections, while 14 cases (36.8%) involved coinfections. Viral etiologies were largely predominant, with 34 children (89.5%) having at least one confirmed or probable viral agent. Nine children (23.7%) had at least one confirmed or probable bacterial etiology. Tables 2 and 3 summarize the different etiological agents identified in our cohort and for each individual patient, respectively.

Among the 28 children with at least one identified etiology, confirmed diagnoses were established in 14 patients (including 8 coinfections), probable diagnoses in 13 patients (with 14 coinfections), and one patient had only a possible diagnosis.

Clinical outcome was unfavorable in 12 children (31.6%), requiring admission to intensive care. Indications included status epilepticus, severe neurological impairment, a Glasgow score <8, hemodynamic instability, brainstem involvement, acute hydrocephalus, decerebrate posturing, and healthcare-associated infections.

Nine children (23.7%) died. Neurological sequelae were documented in 9.7% of survivors, including psychomotor delay, epilepsy, behavioral disorders, memory impairment, intellectual disability, relapse, and eating disorders. The median length of hospital stay was 19.5 days (range: 4–874 days).

Table 1. Epidemiological, clinical, biological, etiological, and outcome characteristics of children with encephalitis / meningoenkephalitis (n = 38).

Variable	n	%
Male sex	22	—
Sex ratio (M/F)	1.37	—
Comorbidities	4	10.5
Immunosuppressive or corticosteroid therapy	3	7.9
Winter season	—	47.4
Abrupt onset	31	81.6
Altered consciousness	37	97.3
Glasgow score < 8	7	18.4
Behavioral disturbances	13	34.2
Focal neurological deficits	22	57.8
Seizures	28	73.7
Respiratory involvement	14	36.8
CSF pleocytosis (n=37)	31	83.8
Elevated CSF protein (n=37)	19	51.4
Hypoglycorrhachia (n=37)	12	32.4
Abnormal neuroimaging	23	60.5
Abnormal EEG (n=22)	9	40.9
Admission to intensive care	12	31.6
Viral etiology (confirmed/probable)	34	89.5
Bacterial etiology (confirmed/probable)	9	23.7
Etiology not identified	10	26.3
Deaths	9	23.7
Neurological sequelae	3	9.7

Table 2. Identified etiological agents in children with encephalitis/meningoenkephalitis (n = 38).

Etiological agent	Patients (n)	Confirmed	Probable	Possible
HSV-1	1	0	1	0
Varicella-zoster virus	2	0	2	0
<i>Mycoplasma pneumoniae</i>	3	0	1	2
<i>Chlamydiae</i> spp.	2	0	0	2
Tuberculosis (<i>Mycobacterium tuberculosis</i>)	3	1	0	2
Enterovirus	2	1	1	0
Epstein–Barr virus	7	3	4	0
Cytomegalovirus	3	1	2	0
HHV-6	1	0	1	0
Adenovirus	4	2	2	0
<i>Borrelia burgdorferi</i>	2	0	0	2
Rubella virus	1	0	1	0
Influenza A	2	0	2	0
Influenza B (Yamagata lineage)	1	0	1	0
Parvovirus B19	1	0	1	0
<i>Streptococcus pneumoniae</i>	3	3	0	0
<i>Neisseria meningitidis</i>	3	3	0	0
<i>Klebsiella</i> spp.	1	1	0	0
Coronavirus	8	0	8	0
Rhinovirus	1	0	1	0
Total diagnoses	51	15	28	8

Coinfections included.

Table 3. Confirmed etiological agents identified per patient (including coinfections).

Etiological agent	Confirmed n (coinfections)	Probable n (coinfections)	Possible n (coinfections)	Total (%)
Tuberculosis	1 (CMV, coronavirus)	—	1	2 (5.3)
Epstein–Barr virus	3 (coronavirus, HSV-1, TB)	1	—	4 (10.5)
<i>Streptococcus pneumoniae</i>	3 (enterovirus, mycoplasma)	—	—	3 (7.9)
Cytomegalovirus	1	1 (EBV)	—	2 (5.3)
Adenovirus	2	2 (HHV-6, parvovirus B19)	—	4 (10.5)
Enterovirus	—	1 (rhinovirus)	—	1 (2.6)
<i>Neisseria meningitidis</i>	3	—	—	3 (7.9)
Rubella virus	—	1	—	1 (2.6)
<i>Klebsiella</i> spp.	1	—	—	1 (2.6)
Varicella-zoster virus	—	2 (Borrelia, mycoplasma, chlamydiae)	—	2 (5.3)
Coronavirus	—	2 (EBV, chlamydiae, mycoplasma)	—	2 (5.3)
Influenza A	—	2 (EBV)	—	2 (5.3)
Influenza B	—	1 (coronavirus)	—	1 (2.6)
Total patients	14	13	1	28 (73.7)

4. DISCUSSION

Among the 141 patients included in the study, 38 children met the diagnostic criteria for encephalitis or meningoencephalitis, representing 27% of the total cohort. Neurological impairment was prominent in this pediatric population, with altered consciousness observed in 37 children (97.3%), behavioral disturbances in 13 (34.2%), focal neurological deficits in 22 (57.8%), and seizures in 28 (73.7%). Cerebrospinal fluid pleocytosis was present in 31 of 37 children who underwent lumbar puncture (83.8%). Abnormal neuroimaging findings were reported in 23 cases (60.5%), and electroencephalographic abnormalities compatible with encephalitis—non-mutually exclusive categories—were observed in 22 of 38 children (40.9%). Most children reported having lived in urban or rural areas with high population density during the three months preceding symptom onset (50% and 47.4%, respectively). For some patients, both blood and cerebrospinal fluid samples were tested for more than 28 pathogens.

The proportion of pediatric cases (27%) was comparable to that reported in a study based on hospitalization discharge summaries recorded in the French national hospital database (PMSI), in which childhood encephalitis accounted for 29% of cases (26, 27). In our cohort, a male predominance was observed, with a sex ratio of 1.37, along with a slight winter predominance. By comparison, the PMSI-based study reported a lower male predominance (sex ratio 1.2), no seasonal variation, and a markedly lower case-fatality rate (2.3% versus 23.7% in our study) (26, 27).

In Batna, a wide spectrum of pathogens was responsible for acute encephalitis, as shown in tables 2 and 3. Using a comprehensive diagnostic approach and advanced laboratory techniques, an etiological diagnosis was established in 28 children (73.7%). This diagnostic yield was higher than that reported in studies from Japan (1983–1990; 256 cases) (28) and Greece (2005–2009; 42 cases) (29), where causative agents were identified in only 41% and 57% of cases, respectively. By contrast, our results were comparable to those reported in studies from Saint-Étienne, France (1991–2002; 32 cases) (30) and Slovenia (1979–1991; 170 cases) (31), with etiological identification rates of 81% and 68%, respectively.

A monomicrobial infection was identified in 14 patients (36.8%). We also observed a number of coinfections; however, due to multiple positive results, the complexity of interpretation increased. Such complex coinfections have also been reported by others, with rates of 2% in Great Britain (32) and 38.9% in Thailand (33)

In our cohort, the etiology remained unidentified in 10 children (26.3%), a proportion substantially lower than that reported in Japan (59%), Greece (43%), and Slovenia (32%)(31), but higher than that observed in Saint-Étienne (19%)(30). When coinfections were taken into account, viral etiologies predominated, led by coronavirus (21.1%), followed by Epstein–Barr virus (18.4%), adenovirus (10.5%), and influenza virus (7.9%). Among bacterial causes, *Streptococcus pneumoniae* (7.9%), *Neisseria meningitidis* (7.9%), and *Mycobacterium tuberculosis* (BK; 5.3%)—which is endemic in Algeria—were the most frequently identified. These findings contrast with those of the PMSI study, in which varicella-zoster virus, herpes simplex virus, and arboviruses were the most commonly reported agents (34).

Marked geographical variability in etiological patterns has been documented. (*cf.* Table 4). In Finland between 1973 and 1987, varicella-zoster virus was the most frequently diagnosed pathogen (25%), followed by mumps (8%), herpes simplex virus (7%), and measles virus (4%)(24, 35). In Slovenia (1979–1991), 68% of children had an identified etiology (24, 31), with tick-borne encephalitis

as the leading cause (29%), followed by varicella-zoster virus (17%), herpes simplex virus (10%), rubella virus (3%), mumps virus (3%), and measles virus (1%), reflecting the epidemiological specificity of a region where a particular arbovirus is highly prevalent. In Japan (1983–1990), a multicenter pediatric study involving 36 hospitals reported measles virus (23%)(24, 31), herpes simplex virus (20%), and rubella virus (23%) as the most frequent etiologies; the concurrent rubella epidemic likely led to an overestimation of its role, and the study period preceded the implementation of nationwide measles eradication programs.

Similarly, a study conducted in a pediatric intensive care unit in Saint-Étienne, France (1991–2002), identified varicella-zoster virus (31%), herpes simplex virus (19%), and enteroviruses (13%) as the most common causes (24, 30). In Greece, a multicenter prospective study identified an etiology in 24 of 42 children (57%), with herpes simplex virus (42%) and enteroviruses (25%) predominating(29).

Overall, our findings highlight considerable heterogeneity in the etiological distribution of pediatric encephalitis across regions and time periods.

Table 4. Comparison of identified etiological agents in pediatric encephalitis and meningoencephalitis across international studies.

Etiology	Batna 2012–2015 n=38 (%)	Japan 1990 (%)	Japan 1983–1990 n=256 (%)	Saint-Étienne (France) 1991–2002 n=32 (%)	PMSI (France) 2002 (%)	Slovenia 1979–1991 n=170 (%)	Greece 2005–2009 n=42 (%)
Encephalitis without identified etiology	10 (26.3)	59		19	—	32	43
Identified etiology	28 (73.7)	41		81	—	68	57
Herpes simplex virus	1 (2.6)	20		19	—	10	42
Varicella-zoster virus	2 (5.3)	—		31	—	17	—
Enterovirus	2 (5.3)	—		13	—	—	25
Epstein–Barr virus	7 (18.4)	—		—	—	—	—
Cytomegalovirus	3 (7.9)	—		—	—	—	—
Adenovirus	4 (10.5)	—		—	—	—	—
HHV-6	1 (2.6)	—		—	—	—	—
Rubella virus	1 (2.6)	23		—	—	3	—
Measles virus	0	23		—	—	1	—
Mumps virus	0	—		—	—	3	—
Parvovirus B19	1 (2.6)	—		—	—	—	—
Coronavirus	8 (21.1)	—		—	—	—	—
Influenza A	2 (5.3)	—		—	—	—	—
Influenza B	1 (2.6)	—		—	—	—	—
Rhinovirus	2 (5.3)	—		—	—	—	—
Tick-borne encephalitis virus	0	—		—	—	29	—
<i>Mycobacterium tuberculosis</i>	3 (7.9)	20		19	—	10	42
<i>Chlamydiae</i> spp.	2 (5.3)	—		31	—	17	—
<i>Mycoplasma pneumoniae</i>	3 (7.9)	—		13	—	—	25
<i>Borrelia burgdorferi</i>	2 (5.3)	—		—	—	—	—
<i>Streptococcus pneumoniae</i>	3 (7.9)	—		—	—	—	—
<i>Neisseria meningitidis</i>	3 (7.9)	—		—	—	—	—
<i>Klebsiella</i> spp.	1 (2.6)	—		—	—	—	—

Finally, mortality in our pediatric cohort was high, with 9 deaths (23.7%), compared with a case-fatality rate of 2.3% reported in the PMSI 2002 study. Neurological sequelae were documented in 3 children (9.7%), underscoring the severity of encephalitis and meningoencephalitis in this population and the need for early diagnosis and targeted management.

Lethality risk factors identified in our study were hospitalization in the ICU ($p < 10^{-3}$), Glasgow Coma Scale < 8 , presence of *rhinovirus* ($p = 0.032$), and tuberculosis ($p = 0.030$). Our results differed considerably from those observed in the study conducted in France (25) where a fatal outcome occurred in only 26 patients (10%) during hospitalization.

It was essential that this study be based on the existing infrastructure; the additional analyses performed at the Lyon laboratory ultimately served only to better document, from a microbiological perspective, the cases observed in Batna after the fact.

Furthermore, the absence of certain examinations in some hospital facilities (such as imaging or EEG) hindered rapid etiological diagnosis and thus optimized patient management.

To our knowledge, this is the first prospective study of this scale conducted in Algeria and Africa, with a sample size comparable to those carried out for similar objectives in the United States, the United Kingdom, or France. From an etiological perspective, our approach revealed a high number of possible "co-infections." Some of these merely reflect the reactivation of endogenous viruses, whose responsibility in the infectious process was difficult to determine. The observed differences also highlight the specific features of Algerian epidemiology and therefore the need for an approach tailored to the country's situation, in terms of the incidence of different pathogens and the optimized management that should result.

5. CONCLUSION

This study provides the first comprehensive description of the etiological spectrum of pediatric encephalitis and meningoencephalitis in eastern Algeria. Early identification of specific etiologies requiring urgent treatment remains crucial to reduce mortality and long-term morbidity. Advances in PCR-based diagnostic techniques, including multiplex assays, offer promising perspectives for rapid and accurate diagnosis, provided that sensitivity improves and costs remain acceptable.

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