Background. In 2014, a global outbreak of Enterovirus D68 (EV-D68) caused severe respiratory disease and was associated with an increase in acute flaccid myelitis (AFM) cases. Despite heightened surveillance, both EV-D68 detection and AFM reporting dropped in 2015. As AFM reporting increased in 2016, we sought to better understand AFM and EV-D68 epidemiology at our institution.

Methods. Chart review of clinical presentation and workup was conducted on patients meeting the case definition for AFM for 2015-16. To determine EV-D68 prevalence at CHLA, samples positive for Rhinovirus/Enterovirus (RV/EV) by FilmArray® Respiratory Panel (FA-RP) in September 2016 were screened for EV-D68 by RT-PCR. **Results** were compared with a research algorithm developed within the FilmArray® Trend epidemiology software. After establishing accurate EV-D68 prediction, the algorithm was used on historic FA-RP assays to measure EV-D68 prevalence at CHLA in 2015 and 2016.

Results. 7 patients with a median age of 3.3 years and no significant past medical history presented with AFM between July 15 - October 15, 2016, while none were identified in 2015. All had acute onset patchy weakness involving mostly the upper limbs and grey matter involvement on MRI. 6/7 reported fever/upper respiratory infection prior to AFM onset. CSF from 7/7 was negative by FilmArray® meningitis/encephalitis Panel and 2/7 were positive for EBV DNA. Further work up on CSF and blood were negative. 4/7 (57.1%) patients were RV/EV positive from respiratory samples and 3 were confirmed as EV-D68 by RT-PCR. IVIG was given in 7/7 cases. Patients were discharged after an average of 8.8 (4.8-13.6) days. The FilmArray Trend monitoring revealed that during the time of AFM presentation in 2016, 226/778 patients tested for respiratory viruses by the FA-RP were positive for RV/EV. Of those, 29.2% (66/226) were positive for EV-D68 compared with 0.02% (2/224) over the same period in 2015.

Conclusion. As shown by CDC surveillance data, we saw a resurgence of AFM cases in 2016 compared with 2015. All 7 patients identified were previously healthy and had persistent weakness at discharge. Cases were accompanied by increases in circulating respiratory EV-D68. Further investigation of the correlation between EV-D68 resurgence and AFM is warranted.

Disclosures. **L. Meyers**, BioFire Diagnostics: Employee, Salary. **J. Jones**, Biofire Diagnostics LLC: Employee, Salary. **J. Dien Bard**, BioFire: Consultant and Investigator, Research grant and Speaker honorarium.

1012. Hearing Loss in Cryptococcal Meningitis Survivors

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Background. Hearing loss is a known complication cryptococcal meningitis (CM); however, there is a paucity of data. We aimed to describe hearing loss in CM survivors.

Methods. We assessed hearing via audiometry 8 and 18 weeks after diagnosis of CM in Kampala, Uganda from 2015-2016. We measured at 0.5, 1, 2, 4 Hz. Normal hearing was defined as minimum hearing level at <25 decibels (dB), mild at 25-39, moderate hearing at 40–69, severe at 70–89, and profound hearing loss at 90+ dB. We compared clinical factors, fungal burden, and CSF parameters to evaluate for factors associated with improvement (change in hearing loss category).

Results. We evaluated hearing symptoms via audiogram at week 8 (n = 117) and week 18 (n = 98). At 8-weeks, 6 (5%) participants had normal hearing, 36 (31%) had mild hearing loss, 72 (62%) had moderate hearing loss, 3 (3%) had severe hearing loss and none had profound hearing loss. Of those with moderate/severe/profound loss at week 8, 63 (54%) had mixed conductive + sensorineural hearing loss, 15 (13%) had sensorineural hearing loss, and 14 (12%) had conductive hearing loss. An additional 19 (16%) had sensorineural loss but unknown air conduction, and 3 (3%) did not have sensorineural loss but unknown air conduction. We compared risk factors for hearing loss summarized in Table 1. We assessed 66 participants who had repeated audiograms at week 8 and week 18. Of those 31 (47%) had no change, 30 (45%) had improvement and 5 (8%) had worsening.

Conclusion. Moderate/severe hearing loss was common 8 weeks after diagnosis of CM. More than half had mixed hearing loss and 20% had conductive hearing loss which represents a higher incidence than noted in other types of meningitis. The data is complicated by advanced HIV. Further research is needed evaluating immunologic factors causes hearing impairment in those who survived CM.

Table 1. Risk Factors for Hearing Loss 8 weeks post Cryptococcal Meningitis.

CSF Parameter	N	Normal Hearing or Mild Hearing Loss	Moderate, Severe, or Profound Hearing Loss	P value via Chi-square
Diagnosis Opening Pressure >25 cm H_2O	113	24 (71%)	28 (45%)	*0.017*
Average Opening Pressure >20 cm H_20	96	34 (81%)	43 (61%)	*0.025*
Quantitative Culture >100,000 CFU/mL	116	14 (33%)	26 (35%)	0.84
Diagnosis CSF WBCs >4 /mL	105	16 (42%)	29 (43%)	0.91

Disclosures. **All authors:** No reported disclosures.

1013. Infectious Causes and Infectious Mimics of Acute Encephalitis: a Prospective Study from Thailand

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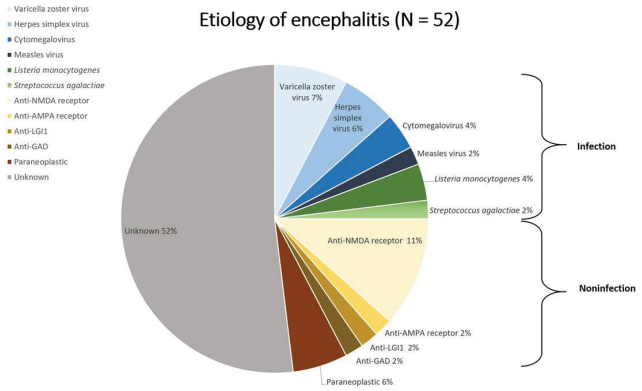
Background. Previous reports of infectious encephalitis in Thailand showed viruses as major pathogens similar to worldwide data. Major viruses in studies varied among Japanese encephalitis, Enteroviruses and Herpesviruses. Infectious etiologies vary by regions, seasons and preventive strategies done. Dynamic change of pathogen is believed to occur continually. Local data in each region is important to develop an algorithm of investigations for the cost-effectiveness.

Methods. This is a prospective study of patients with encephalitis between January 2014 to March 2017 at a tertiary hospital in Bangkok. Microbiological and serological studies were done according to an algorithm based on initial cerebrospinal fluid analysis. Initial tests were for bacteria, fungus, mycobacterium and commonly prevalent viruses. Further tests for infectious etiology were done by stepwise approach if initial tests yielded negative.

Results. Fifty-two patients were enrolled. Twenty-seven (51.9%) patients had no etiology identified. Three patients (5.8%) had bacterial etiology, 10 (19.2%) had viral etiology, and 12 (23%) had immune-mediated encephalitis. Among viral etiologies, VZV was identified in 4 cases, HSV in 3 cases, CMV in 2 cases and measles in 1 case. Baseline characteristic of HIV infection or skin rash was associated with viral infection (p 0.031, p 0.006). Patients with VZV encephalitis might not have active skin lesion. The presence of prodrome, duration of prodrome, neurological onset to peak and physical examination of focal neurodeficit, meningeal irritation signs, and reflex were similar across all etiologies. White blood cell [mean 7.0 (range 0–30) cells/μL] and protein [mean 32.5 (range 11–70.4) mg/dL] from the cerebrospinal fluid of noninfectious etiologies tended to be lower than the levels of infectious causes (p 0.009, p 0.020). All patients survived at 7 days after admission.

Conclusion. A quarter of patients presenting with acute encephalitis in this study had autoimmune and paraneoplastic encephalitis. Infections caused by herpesviruses was the most prevalent viral etiology. Autoimmune and paraneoplastic encephalitis should be kept in the differential diagnosis in patients with acute encephalitis.



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1014. Long-term Outcomes of Acute Aseptic Encephalitis In Adults - a Single Center Study

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Background. Encephalitis is a heterogeneous syndrome associated with significant mortality and neurophysiological sequelae. The etiology is identified in only 20–50% of cases, and long-term outcomes of survivors are underinvestigated, especially in patients with unknown etiology. The aim of this study was to describe long-term outcomes of patients with aseptic encephalitis of various etiologies.

Methods. The study population consisted of a retrospectively identified cohort of consecutive adult patients diagnosed with viral and etiologically undiagnosed encephalitis during a 24-month period (2014-2015) at the University Hospital for Infectious Diseases Zagreb, Croatia. Clinical, laboratory data and short-term outcomes were