**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.** Seven 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 EV DNA. Further work up on CSF and blood were negative. 4/7 (57.1%) patients were RV/EV positive from respiratory samples and 3/7 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 frame presentation of 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.

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1012. Hearing Loss in Cryptococcal Meningitis Survivors Sarah Loften, MD;1 Martha Montgomery, MD, MHS;1 Nathan Yueh, BA;1 Alice Namudde, Nursing;2 Joshua Rhein, MD;3 Mahsa Abassi, DO;2 Abdu Musubire, MMEd;2 David Meyers, PhD,3 David Boulware, MPH, MPH;4 and ASTRO-CM Team;4 1Department of Medicine, Division of Infectious Diseases and International Medicine, University of Minnesota, Minneapolis, Minnesota, 2University of Minnesota, Minneapolis, Minnesota, 3Infectious Disease Institute, Kampala, Uganda, 4Division of Infectious Diseases and International Medicine, Department of Medicine, University of Minnesota, Minneapolis, Minnesota, 4Infectious Disease Institute, Makerere University, Kampala, Uganda

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**Background.** Hearing loss is a known complication in 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. We included patients survived at 7 days after admission. Normal hearing was defined as minimum hearing level at <25 decibels (dB), mild at 25-29, moderate at 40-69, severe at 70-89, and profound hearing loss at >90 dB. We compared clinical factors, funnel burden, and CSF parameters to evaluate 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 (5.3%) 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. At 18-weeks, 2 (2%) 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. An additional 19 (16%) had sensorineural hearing 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. 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 H2O | 113 | 24 (71%) | 28 (45%) | 0.017 |
| Average Opening Pressure >20 cm H2O | 96 | 34 (81%) | 43 (81%) | 0.025 |
| Quantitative Culture >100,000 CFU/mL | 116 | 14 (33%) | 26 (35%) | 0.84 |
| Diagnosis CSF WBC >20 μL | 105 | 16 (42%) | 29 (42%) | 0.91 |

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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 25 (23%) had immune-mediated encephalitis. Among viral etiologies, HSV was identified in 4 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 prorome, duration of prorome, 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 encephalitis 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 herpetic virus 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
collected from medical records, and long-term outcomes were assessed by telephone interviews and quantified through modified Rankin scores (mRS).

**Results.** A total of 90 patients were identified (57.7% female; 51.5 ± 17.4 years). Viral encephalitis was identified in 20 (22.2%) patients: herpes simplex virus (HSV-1, 8.9%), varicella-zoster virus (VZV, 6.7%), Tick-borne encephalitis (TBE, 4.4%) and enterovirus (2.2%). Postinfectious meningocencephalitis was suspected in 14 (15.6%) patients, and 56 (62.2%) had unknown etiology. Elevated CSF WBC was present in 77 patients (mean of 169.3 ± 279.4/mm3) and all but 6 had elevated CSF proteins (1.23 ± 0.88 g/L). Convolutions occurred more frequently in HSV-1 (37.5%) and in unknown etiology group (15.7%). GOS-3 was noted in 50% of HSV, 33% of VZV, 25% of TBE and 24% of unknown group patients during hospitalization. Sexual menigitis was necessary in 17.1% of patients with unknown and 23.5% with viral etiology for the mean duration of 1.8 ± 6.7 and 3.2 ± 6.3 days, respectively. The mean length of stay was 23.2 ± 18.5 days. In hospital mortality was 7.8%. Among 64 survivors, testing were available for follow-up interviews (mean follow-up of 28.6 ± 6.8 months), 73.1% with unknown and 90.9% with viral etiology had favorable outcomes (mRS 0–1); 4 (6.25%) had moderate (mRS 3) and 3 (4.6%) had severe neuropsychological deficits (mRS 4–5).

**Conclusion.** Although the etiology of aseptic encephalitis is often unknown, long-term outcomes are favorable in the majority of patients.

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1016. Implementation of In-house PCR Testing for Herpes Simplex Virus Encephalitis: Impact on Patient Acyclovir Exposure

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**Background.** HSV encephalitis is a life-threatening disease process that requires prompt treatment. Diagnosis is often dependent upon nucleic acid studies of CNS samples. We introduced the Film Array Meningitis/Encephalitis (ME) Panel, a qualitative multiplexed nucleic acid-based diagnostic test for a variety of CNS pathogens, including HSV, in June 2016. It replaced an equivalent reference laboratory nucleic acid amplification test. The intent was to provide a more timely result and decrease unnecessary drug exposure to the patient.

**Methods.** We conducted a retrospective chart review of adult patients (≥18 yo) admitted between June September 2015 and June–September 2016 who underwent CSF testing for HSV and received empiric acyclovir. The aim was to determine whether the newly available test resulted in fewer doses of acyclovir in those who tested negative for a herpes virus as compared with those tested with the previously available assay. We excluded those found to be positive for HSV-1, HSV-2, or VZV in the CSF. We defined a dose as any administration of acyclovir or valacyclovir to the patient.

**Results.** Results of the diagnostic assay returned significantly faster after institution of the ME panel (4.9 days (118 hours) vs .9 days (2.25 hours)). Due to the non-normal distribution of acyclovir dosing, comparisons were made with the Wilcoxon Rank-sum. The institution of the ME panel for all CSF studies significantly impacted our use of acyclovir, as patients with negative tests for HSV PCR in the CSF received fewer doses of acyclovir after implementation of the ME panel (median (25th– 75th percentiles) 6 (2–13.5) vs 3 (1–6), P = .018).

**Conclusion.** Implementation of a rapid diagnostic PCR test for HSV 1 and HSV 2 in 2016 significantly reduced the number of empiric acyclovir doses administered during the summer of 2016 compared with the summer of 2015 in patients testing negative for the condition.

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1017. A Primary Amoebic Meningoencephalitis Case Associated with Rafting on an Artificial Whitewater River

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**Background.** Naegleria fowleri is a climate-sensitive thermophilic amoeba found in freshwater that causes primary amoebic meningocencephalitis (PAM; 0–8 infections per year in the U.S.) which enters the nose and migrates to the brain. Patient exposure to water containing the amoeba typically occurs in warm freshwater lakes and ponds during recreational water activities. In June 2016, an 18-year-old woman died of PAM after traveling to North Carolina, where she participated in whitewater rafting on an artificial whitewater river.

**Methods.** To determine water exposures, we reviewed medical records and conducted interviews with family and individuals who had traveled with the case-patient. To further investigate the artificial whitewater river as a possible exposure source, we visited the whitewater facility and collected water, biofilm, and sediment samples from the facility and from the nearby natural river. We performed select water quality tests onsite and tested for the presence of N. fowleri by culture and real-time PCR in the laboratory.

**Results.** Interviews revealed that the case-patient’s most probable water exposure in the 10 days before becoming ill occurred while rafting on an artificial whitewater river during which she was thrown out of the raft and submerged underwater. The ~11.5 million gallons of water in the whitewater facility were filtered, subjected to UV light, and occasionally chlorinated. Heavy algal growth was noted. The free chlorine residual was 0.05 mg/L, turbidity was 6.7 NTU, and water temperature was 30°C in