

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 etiology 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 enteroviruses (2.2%). Postinfectious meningoencephalitis 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/mm<sup>3</sup>) and all but 6 had elevated CSF proteins (1.23 ± 0.88 g/L). Convulsions 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. Mechanical ventilation 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 who 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.

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

#### 1015. Meningitis in the Kaiser Permanente Northern California (KPNC) Pediatric Population 2012 through 2016 and Its Association with Prior Traumatic Brain Injury (TBI)

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**Background.** The incidence of meningitis, particularly bacterial meningitis, is decreasing due to widespread vaccine use with etiologies still due to relatively common organisms. However, risk factors for the development of meningitis are largely unknown. We describe the epidemiology of meningitis among the pediatric population of KPNC and assess the association between prior TBI and meningitis.

**Methods.** We conducted a retrospective descriptive study among KPNC pediatric patients (1 month–18 years) between July 2012 - June 2016 with an ICD-9 meningitis diagnosis. Charts were reviewed to a.) verify and classify the meningitis diagnosis and b.) ascertain prior TBI history. Cases were classified as confirmed, probable, or possible based on clinical course, CSF culture, PCR, and CSF profile. The proportion of meningitis cases with a history of TBI was compared with the published rate of TBI in the general pediatric population.

**Results.** Of 266 cases reviewed, 146 cases were verified as meningitis with 19 confirmed/probable bacterial [(*S. pneumoniae* (7), *E. coli* (3), *S. Agalactiae* (3), *N. meningitidis* (2), *S. Aureus* (1), and not otherwise specified (3)] and 50 confirmed/probable viral [(enterovirus (44), HSV2 (4), HSV1 (1), and WNV (1)] etiologies. An additional 77 possible cases included primarily viral etiologies (65) and a minority of possible bacterial (6) or infection-related inflammatory (6) etiologies. 120 were excluded. The overall meningitis and confirmed bacterial meningitis estimated incidence rate was 17.9 per 100,000 and 2.7 per 100,000, respectively. Twenty meningitis cases (13.7%) had a history of TBI (all of which were concussions) compared with the concussion rate in the general pediatric population reported by AL Zhang et al of 0.7% ( $P < 0.001$ ).

**Conclusion.** Our results are consistent with recent studies showing a decline in bacterial meningitis. Enteroviral meningitis remains the leading cause of identified etiologies. However, in a sizable proportion of cases there was no specific pathogen identified. An increased risk of developing meningitis may be associated with TBIs.

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

#### 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.

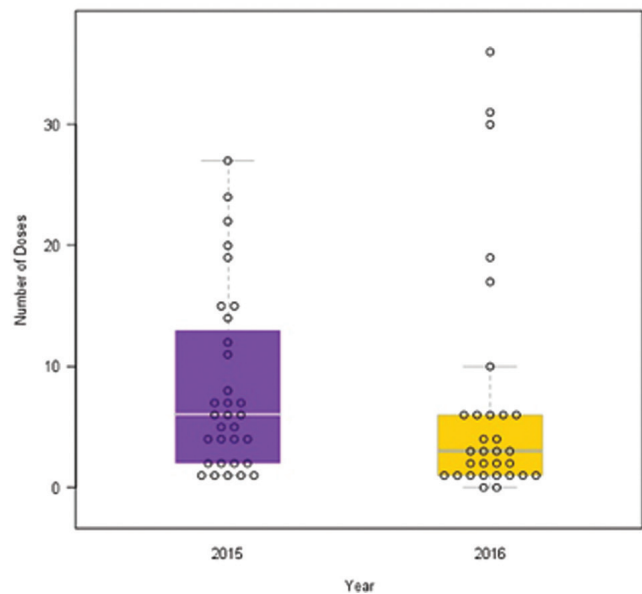
**Methods.** We conducted a retrospective chart review of adult patients (>18 yo) admitted between June-September 2015 and June-September 2016 who underwent CSF testing for HSV and received empiric acyclovir. The aim was to determine whether

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 .09 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 (25<sup>th</sup>–75<sup>th</sup> percentiles) 6 (2–13.5) vs 3 (1–6),  $P = .018$ ).

**Conclusion.** Implementation of a rapid diagnostic PCR based 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.

Doses of Acyclovir by Year



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

#### 1017. A Primary Amebic 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 amebic meningoencephalitis (PAM; 0–8 infections per year in the U.S.) when it 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

the whitewater facility during the site visit. All 11 water-related samples taken from the facility were positive for *N. fowleri*. Of 5 samples taken from the natural river, 1 sediment sample was positive for *N. fowleri*.

**Conclusion.** This investigation documents a novel exposure to an artificial whitewater river as the likely exposure causing PAM in this case. Conditions in the whitewater facility (warm, turbid water with little chlorine and heavy algal growth) rendered the water treatment ineffective and provided an ideal environment for *N. fowleri* to thrive. The combination of natural and engineered elements at the whitewater facility create a challenging environment to control the growth of *N. fowleri*.

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#### 1018. Lyme Disease in Hispanics in Long Island, New York: A New Health Disparity in the U.S.

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**Background.** Lyme disease (LD) is the most commonly reported vector-borne illness in the U.S. A risk factor for acquiring LD is the exposure to outdoors. In Long Island, Hispanics comprise a large share of the outdoor occupational workforce.

**Methods.** A retrospective chart review was performed in all patients with ICD-9 or ICD-10 diagnostic codes for LD between 2011–2016 in SHH and 2010–2015 in SBUH. Inclusion criteria was defined as a clinical scenario compatible with LD (erythema migrans -EM, arthritis, central nervous system (CNS) involvement, carditis, meningitis) and serological confirmation by western blot according to CDC (SBUH) or Imugen\* (SHH) criteria.

**Results.** Out of 1,026 cases (766 SBUH;260 SHH) that carried a diagnosis of LD, 284 cases (22% Hispanics) met inclusion criteria and were added to final analysis (241 SBUH: 43 SHH). The mean age was 48.8 (SD:17.8) and 48.3 (SD:17.5) years-old in H and NH, respectively ( $p > 0.05$ ). Most were male (H:62.2%; NH: 54.3%;  $P = 0.2$ ). In the univariate analysis, headaches were more frequently present in H (42.6%) than in NH (26.6%) ( $P = 0.015$ ). In the logistic regression analysis, the following symptoms were significantly different between H and NH: headaches (OR 1.17, 96% CI 1.60–6.59,  $P = 0.001$ ) and peripheral neuropathy (OR 0.38, 95% CI: 0.15–0.96,  $P = 0.04$ ). Among seasons, H were more frequently diagnosed with Lyme during spring months than NH (H: 26.3% vs. NH: 12.6%,  $P = 0.03$ )

**Conclusion.** Hispanics have a greater risk for presenting with headaches and less peripheral neuropathy than NH. Initiation of outdoor work among H may be the reason of this disproportionate presentation of LD symptoms during spring months. These findings may suggest the propensity for Hispanics to go underdiagnosed with LD, despite their high likelihood of exposure through their occupations in this region.

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#### 1019. Varicella-zoster virus Neurovasculitis (VZV-NV) in the Setting of Autoantibodies to Interferon alpha (anti-IFN $\alpha$ )

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**Background.** VZV-NV resulting in CNS damage is rare in immunocompetent hosts. Humoral immunity protects against acquiring VZV, and T-cell mediated immunity shields against reactivation of the latent virus. Individuals who are immunocompromised due to T-cell mediated defects can present with systemic VZV.

**Methods.** Case report of a previously healthy man who developed VZV-NV associated with autoantibodies to IFN $\alpha$ .

**Results.** A previously healthy 64 year-old man developed an acute T3 Brown-Sequard syndrome. Symptoms progressed to include bilateral lower extremity weakness, ptosis, ophthalmoplegia, and encephalopathy. Magnetic resonance imaging (MRI) showed diffuse T2 hyperintensities with enhancement throughout the spine and brain, including enhancement of his meninges, roots, and cranial nerves. Successive cerebrospinal fluid (CSF) studies revealed increasing B-cell lymphocytosis, (maximum 661 cells/ml), and CSF protein (maximum 242 mg/dL). CSF PCR was positive for VZV and IgM antibodies. Further testing showed anti-IFN $\alpha$  autoantibodies in the plasma and CSF. Serum anti-IFN $\alpha$  fluorescence intensity was 30 times normal, and his plasma blocked IFN $\alpha$ -induced STAT-1 phosphorylation in normal monocytes. Treatment with acyclovir and methylprednisolone resulted in improvement. Repeat LP following treatment revealed 32 WBC/ml with normal protein. Follow-up MRI did not show any new lesions. Three years after initial presentation,

he continues to be stable without clinical relapses, or subclinical changes on MRI. Serum studies were positive for VZV IgG and negative for IgM. CSF PCR was positive for VZV. Lastly, serum anti-IFN $\alpha$  fluorescence intensity remained 25 times normal, and his plasma continued to block IFN $\alpha$ -induced STAT-1 phosphorylation in normal monocytes.

**Conclusion.** This is the first identified case of CNS VZV-NV in the setting of binding and blocking autoantibodies to IFN $\alpha$  in the serum and CSF. Elevated serum and CSF levels of anti-IFN $\alpha$  may impair natural killer, T-cell and neuronal antiviral activity. Disruption of T-cell mediated immunity due to anti-IFN $\alpha$  could cause an adult-onset immunocompromised state with severe VZV reactivation. The association of high-titer neutralizing autoantibodies to IFN $\alpha$  in an adult with CNS VZV is novel and may be clinically relevant.

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#### 1020. Development of a Novel Anthrax Vaccine Comprising LF-PA Chimera

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**Session:** 138. Adult Immunization - Miscellaneous

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**Background.** *Bacillus anthracis* (BA), the etiological agent of anthrax, secretes protective antigen (PA), lethal factor (LF), and edema factor (EF) as major virulence factors. Among them, PA based vaccines are most indispensable for providing immunity against BA, but the low shelf life limits its reliability. Previous studies revealed that PA domain IV includes B-cell epitopes designated as ID I, ID II, and ID III; among them, ID II and ID III have been found to possess more toxin neutralization activity and produce high antibody titre. Moreover, N-terminal region of both LF and EF carries binding site of PA which are homologous to each other. Here, in this study we have developed and evaluate the vaccine efficacy of chimeric vaccine containing ID II-ID III region of PA and N-terminal region of LF and EF (ID-LFN).

**Materials and Methods.** ID-LFN was generated by overlapping PCR followed by cloning in pET28a. The recombinant protein was then expressed and purified by Ni-NTA chromatography. Reactivity of ID-LFN with anti-PA/LF/EF antibodies was checked by ELISA. Stability was assessed using Circular Dichroism Spectroscopy. The vaccine potential of ID-LFN was evaluated by toxin neutralization assay, lymphocyte proliferation assay, and cytokine analysis. The protection efficacy was analyzed by challenge studies in mice.

**Results.** ID-LFN was found to be significantly stable as compared with protective antigen. Anti-ID-LFN antibodies recognized PA, LF as well as EF. Though, the total antibody titre, toxin neutralization activity was found to be less than PA but surprisingly, the protection efficacy of ID-LFN was found similar as PA.

**Conclusion.** The ID-LFN vaccine might be second next generation vaccine showing equal protection but higher shelf life as PA with the capability of neutralizing PA, LF as well as EF at the same time. Thus, it may prove an efficient and reliable treatment strategy against anthrax.

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#### 1021. Phase 1 Clinical Trial of a Replication-Defective Human Cytomegalovirus (CMV) Vaccine

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**Background.** Congenital CMV remains an unmet medical need worldwide. Naturally acquired CMV immunity in women prior to pregnancy has been shown effective in reducing maternal-fetal transmission. V160 is engineered as a replication-defective CMV, and its replication in culture is controlled by a synthetic chemical. V160 can't replicate in humans but it maintains all virological properties for presentation of viral antigens, including gH/gL/pUL128-131 pentameric complex, important for potent neutralizing antibodies (NABs).

**Methods.** Approximately 190 CMV seronegative and seropositive adults at study entry received 3 doses of V160 or placebo administered via intramuscular (IM) or intradermal (ID) route on Day 1, Month 1, and Month 6. Four antigen levels (10, 30, 100, and 250 units per dose) formulated with or without aluminum phosphate adjuvant were evaluated. In each vaccination group, approximately 10 and 4 subjects received study vaccine and placebo, respectively. Injection site and systemic adverse events (AEs) were collected for 14 days after each vaccination. Serious AEs (SAEs) were assessed up to Month 18. Viral shedding (urine and saliva) were monitored up to Month 12. CMV-specific NABs and cell-mediated immune responses (CMI) were measured prior and 1 month after each vaccination, and at Months 12 and 18.

**Results.** During the study, no serious AEs were reported and only one CMV seropositive subject had non-vaccine type viral shedding. In both seronegative and