Conclusion. SSAs are beneficial in the long-term treatment of patients with subarachnoid NCC or chronic perilesional edema. The formulation of guidelines that include multiple options for SSAs will be essential in guiding management of complicated NCC.

Figure 1. T2-weighted MRI images for patient 1 revealed revealed multiple subarachnoid cysts in the basilar, perimesencephalic, quadrigeminal, and perisylvian cisterns.

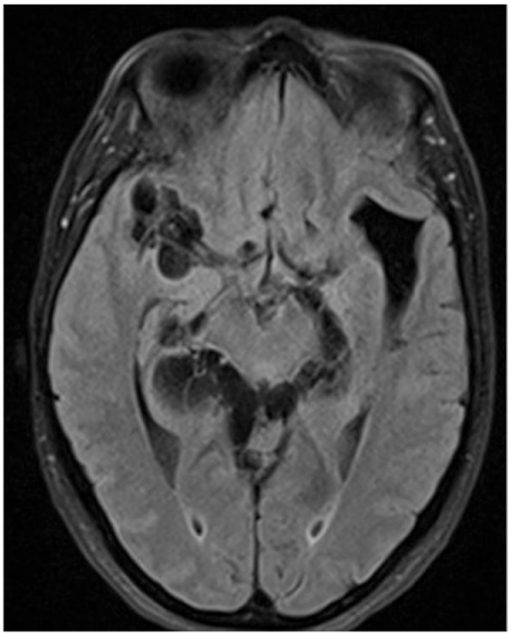


Figure 1. Post-contrast T1-weighted MRI images for patient 2 revealed A) a nodular focus of enhancement within the fourth ventricle with B) resulting obstruction and dilation.

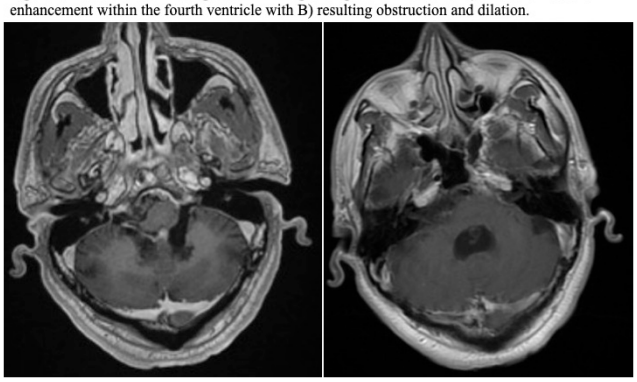


Figure 3. A) T1-weighted post-contrast and B) T2-weighted FLAIR MRI images for patient 3 revealed revealed multiple ring-enhancing, cystic lesions with extensive surrounding edema.

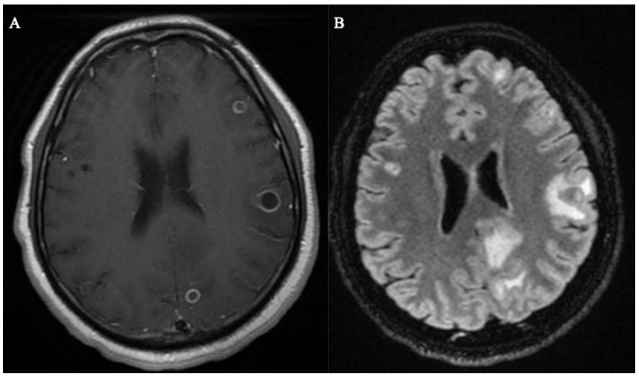
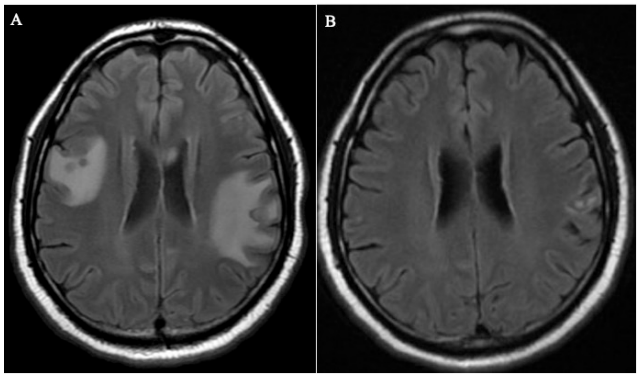


Figure 4. T2-weighted FLAIR images for patient 3 A) on steroids and MTX alone and B) after the addition of adalimumab, demonstrating marked improvement in perilesional edema.



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1407. Potential Impact of the Biofire® Film Array Meningitis and Encephalitis (ME) Panel in Reducing Repeat Lumbar Punctures in Patients with Meningitis and Encephalitis

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Background. The Biofire® FilmArray Meningitis Encephalitis (FAME) is a multiplex polymerase chain reaction (PCR) test can rapidly detect up to 14 pathogens that cause meningitis and encephalitis. The impact on preventing repeat lumbar punctures to obtain more diagnostic studies is currently unknown.

Methods. Patients admitted to Memorial Hermann Hospital (MHH) between July 2018-February 2019 with community-acquired symptoms of meningitis or encephalitis, CSF with white blood cell count >5 cells/mm^3, and with leftover CSF at the MHH microbiology laboratory were eligible for the study. Testing FAME was performed after discharge for specimens that had not been centrifuged, had a volume of ≥200 μL, were appropriately stored, and were collected by lumbar puncture (LP) for evaluation of suspected meningitis/encephalitis.

Results. Of 1,382 CSF specimens screened, 70 (5.0%) met the criteria and were tested with FAME. The majority was adults (72.8%), non-Caucasian (68.6%), male (60%), immunocompetent (75.7%) and had a meningitis presentation (75.7%). Mean age was 36.9 years (1 mo-89 years). The mean duration between CSF collection and any PCR result done in the hospital was 60 hours. Fifteen patients (21.4%) required 25 repeat LPs [13 (86.6%) for additional testing (7 (53.8%) pediatric patients) and 2 (13.3%) for cryptococcal meningitis assessment]. The FAME could have prevented repeat LPs in 86.6% of patients. Five of the 13 repeat LP (38.4%) FA ME showed a pathogen [VZV (2), HSV 1 (1), HHSV-6 (1*), Neisseria meningitidis* (1)]. Of 46 tests with negative FA ME, acyclovir therapy was started in 22 (47.8%) with a mean of 6 doses dispensed. 38 (26.6%) patients were discharged with an unknown etiology of whom FA ME was positive in 8 (21%) [HSV2 (37.5%), VZV (25%), Enterovirus (25%) and HSV1 (12.5%)]. PCR was ordered in the hospital for only 4 (50%) of these patients.

Conclusion. The FAME identified an etiology in 21% of patients with meningitis and encephalitis symptoms discharged with an unknown etiology. A total of 18.5% of patients required a repeat LP for additional testing. FAME testing offers an avenue for reducing the burden of repeat LPs and duration of unnecessary anti-infective therapy while increasing diagnostic yield.

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

1408. *Treponema pallidum*-Specific Antibody Testing in the Evaluation of Neurosyphilis, a Prospective Trial

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Background. The therapeutic challenges of neurosyphilis are rooted in its diagnosis and management, with potential for complications arising from asymptomatic, unrecognized, or under-treated disease. Currently, the non-treponemal VDRL testing of cerebrospinal fluid (CSF) samples is used to predict those with CNS invasion by *T. pallidum*. However, more extensive evaluation of those at any stage of infection demonstrates both that the incidence of CNS invasion is much greater than predicted, and there exists a large proportion of false positives from VDRL testing alone.

Methods. Subjects with suspected neurosyphilis were recruited from the infectious disease clinic after referral to LAC+USC Hospital. Informed consent was obtained and subjects underwent clinical examination, including a standardized neurological and neurocognitive evaluation and CSF sampling. A CSF-specific VDRL, FTAabs, and a *T. pallidum* particle agglutination index were calculated:

A TPPA Index >2.0 was defined as positive and definitive evidence of neurosyphilis

Results. 40 subjects were recruited, 8 were HIV-negative and 32 HIV positive, of which, 1 declined to continue after CSF sampling (Table 1). Employing the CSF TPPA index, 7/31 HIV positive (22.6%) and 1/8 HIV-negative individuals (12.5%) had neurosyphilis (Table 2). Discordant results with the CSF VDRL were common; 4/31 subjects (12.9%) with a positive CSF VDRL had a TPPA Index < 2.0 (0.227, 0.227, 0.315, and 0.400) and 4/31 subjects (12.9%) with a negative CSF VDRL had a positive TPPA index (2.234, 3.333, 3.797, and 4.548, Table 3). Neurocognitive and neurologic abnormalities were commonly encountered in this population both with and without documented neurosyphilis.

Conclusion. Our investigations demonstrate the value of CSF sampling in persons with any stage of syphilis and establish the utility of *T. pallidum*-specific antibody testing to greatly facilitate clinical decision-making. The diagnostic tools to evaluate the *T. pallidum*-specific immunological response of the CNS to syphilis are currently widely available, inexpensive, but woefully underutilized

Table 1. Subject demographics

	HIV-positive	HIV-negative
Subjects	32 (30 male, 2 female)	8 (4 male, 4 female)
Median Age in years (Range)	41 (23 – 62)	44 (27 – 63)
Median RPR titer (Range)	1:64 (1:4 – 1:2048)	1:16 (1:2 – 1:128)

Table 2. Key laboratory values

		HIV-positive Median (Range)	HIV-negative Median (Range)
CSF	WBC (cells/mm^3)	2 (0 – 129)	2 (1 – 11)
	Glucose (mg/dL)	57 (42 – 78)	56 (48 – 93)
	Protein (mg/dL)	25 (10 – 55)	18 (13 – 45)
	Albumin (mg/dL)	12.8 (4.2 – 23.7)	9.9 (7.3 – 16.0)
	CSF VDRL Positive	7	1
	CSF FTA_{abs} Positive	19	1
	TPPA Titer	2 (0 – 200)	0 (0 – 1000)
Serum	Protein (g/dL)	7.4 (6.0 – 8.9)	7.4 (7.2 – 8.4)
	Albumin (g/dL)	4.4 (2.6 – 4.9)	4.5 (4.0 – 5.0)
	HIV Viral Load	1.6 (1.3 – 6.22)	NA
	CD_4 Cell Count	371 (8 – 570)	NA
	TPPA Titer	1000 (100 – 100,000)	1000 (700 – 10.000)

Table 3. Statistical measures in HIV only patients

	True Positive[1]	True Negative[2]	
VDRL Positive	3	4	PPV = 43%
VDRL Negative	4	21	NPV = 84%
	Sensitivity = 43%	Specificity = 84%	

[1]As defined by an antibody titer in excess of 2 times as could be explained by passive diffusion across the blood-brain barrier
[2]As defined by an antibody titer that is less than what could be accounted for by passive diffusion across the blood-brain barrier

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

1409. Next-Generation Sequencing-based Detection of *Angiostrongylus cantonensis* (AC) Using Microbial Cell-free DNA Sequencing of Plasma in Atypical Cases of Rat Lungworm Meningitis Presenting with Ascending Paralysis

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Background. AC or Rat Lungworm meningitis usually presents as a self-limited illness with headache and sensory changes, rarely progressing to coma, death, or permanent brain damage. It is usually diagnosed by eosinophils in the CSF. Once limited to Asia and the tropical Pacific AC transmission via slugs and snails documented on US mainland in 2018. We describe 2 unusual, severe examples of AC infection in infants presenting with ascending paralysis and initial CSF without eosinophilia suggesting Guillain–Barre syndrome (GBS).

Methods. Conventional lab testing of serum and CSF, brain and spine MRI, AC PCR by Hawaii Department of Health, and mcfDNA next-generation sequencing (NGS) of plasma (Karius).

Results. Two infants, aged 8 and 11 months, presented with fever, lower extremity weakness, and ascending paralysis. An initial evaluation in both included normal brain/spine imaging and CSF with modest lymphocytic pleocytosis without eosinophils. Paralysis progressed despite IVIG. Case 1: 11-month male: Admitted on fever day 5. Paralysis progressed to respiratory failure requiring ventilation for 20 days. Illness day 16: MRI showed spinal cord swelling C3-C7, Brain normal. CSF#3: WBC 269 28% eos, ↑ protein. Visible 8 mm long young adult worms, PCR positive for AC. Rx high-dose corticosteroids, albendazole for >4 weeks. Day 29 illness MRI: Cerebral infarct L frontal lobe, worm tracks medulla, inflammation cauda equina. Slow improvement over 5 months. Case 2: 8-month-old female: Admitted fever day 8. Weakness progressed to arms and trunk. Day 10 illness: CSF #2: Visible worms present, WBC 84, 26% eos. PCR positive for AC. Rx: High-dose steroids and albendazole x4 weeks. MRI spine illness day 30: inflammation cauda equina. Weakness improved by illness day 37. McfDNA sequencing of plasma detected AC in acute stage peaks of 123 and 12 molecules/microliter in cases 1 and 2. Serial mcfDNA testing showed a decline in the AC DNA level in plasma which correlated with treatment and clinical response.

Conclusion. AC infection may mimic GBS or transverse myelitis. AC diagnosis may require repeat CSF testing. NGS detection of AC in plasma holds promise as rapid, noninvasive diagnosis and assessment of response to therapy. High-dose steroids with albendazole may be effective even in severe AC.

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1410. Serious Cryptococcal Infections with Ruxolitinib Use: A Case of Meningitis and a Review of the Literature

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Background. Ruxolitinib is an inhibitor of Janus kinase (JAK) 1 and 2 and is approved for the treatment of myelofibrosis and polycythemia vera. Infectious complications associated with its use include reactivation of herpes simplex, zoster, hepatitis B, and tuberculosis, mucormycosis, and progressive multifocal leukoencephalopathy.

Methods. Seven cases of ruxolitinib-associated cryptococcal infections have been reported: three cases of meningitis, two cases of pulmonary disease, and two cases of disseminated disease (Table 1).

Results. We present a 72-year-old male with a history of JAK-2-positive polycythemia vera with secondary myelofibrosis, and concurrent multiple myeloma who presented with 3 weeks of chronic cough and 3 days of fever with severe bifrontal headache after remodeling a large birdcage in his backyard. The patient was on ruxolitinib, ixazomib, and weekly dexamethasone. Cerebrospinal fluid (CSF) analysis showed an elevated opening pressure of 29 cm of CSF, 173 leucocytes with a predominance of lymphocytes, protein of 87 mg/dL and a normal glucose level. BioFire FilmArray® Meningitis/Encephalitis panel detected targets for Cryptococcus in the CSF, and CSF cryptococcal antigen was positive at 1:4. CSF fungal cultures were subsequently positive for Cryptococcus neoformans, susceptible to liposomal amphotericin B (LAMB) and fluconazole. Ruxolitinib was discontinued, treatment with LAMB and flucytosine (5-FC) was associated with significant improvement of headache over the next week. Repeat CSF analysis in 2 weeks was culture negative with a negative cryptococcal antigen test. The patient completed 3 weeks of LAMB and 5-FC and then transitioned to oral fluconazole for a year.

Conclusion. Ruloxitinib and other JAK inhibitors suppress host immunity through impaired T-cell activation and downregulation of cytokines. The JAK-Signal Transducer and Activator of Transcription pathway is one of the most active pathways in host defense against cryptococcus, and use of JAK inhibitors like ruxolitinib may predispose to severe cryptococcal infections. Bird exposure is a risk factor, making environmental contact counseling for patients on ruxolitinib important. Fluconazole prophylaxis needs to be considered in select cases.

Table. Cryptococcal infections associated with Ruxolitinib

Year/ Author	Age Sex	Underlying Dx	Duration of Dx	Length of Therapy	Avian Exposure	Cryptococcal Syndrome	Symptom/ Duration	Diagnosis of Cryptococcal infection	Treatment	Outcomes
2013/Nicholas G. Wysham	66-M	PV MF	12 Y	18 Mo	None	CRP Neoformans Pneumonia	6 weeks of dyspnea, cough, fever	Bronchoscopy With BAL	Fluconazole	Resolution of fever and cough
2015/ Fredy H El Sakr	74-M	MF	NA	13 Mo	NR	Disseminated CRP Neoformans infection	Somnolence, confusion, failure to thrive	Fungal culture of CSF and Blood	Amphotericin Flucytosine, Large volume LP	Improvement to mental status
2015/Chih-Cheng Chen	69-F	MF	NR	3 Y	Chickens	CRP ME	Fever and disoriented consciousness	Fungal culture of CSF	Fluconazole Amphotericin	Improvement to mental status
2017/Liu J1	71-M	CMML	NR	3 Cycles	NR	Fungal Pericardial Tamponade, Pericarditis, CRP Fungemia	Extreme leukocytosis and ARI	Pericardiocentesis, Pericardial Fluid Culture, Blood Culture	Micafungin, Fluconazole	Severe MOF, Expired
2017/Anna Hirano	79-M	MF	6 Mo	6 Mo	None	Pulmonary Cryptococcosis	Pulmonary Nodules	Transbronchial Lung Biopsy	Voriconazole	Pulmonary lesions diminished in size
2017/Diovert i	70-M	MF	NR	3 Mo	NR	Disseminated Cryptococcosis	Encephalopathic kidney and liver injury	Blood Culture	NR	Expired
2018/A.Chakrabarti	60-M	MF	2 Y	NR	None	CRP Meningitis	2-3 Mo headache, visual disturbances, gait abnormalities	CSF antigen	Fluconazole	Symptoms resolved

List of Table Abbreviations:
CRP: Cryptococcus
MF- Myelofibrosis
PV- Polycythemia Vera
LP - Lumbar Puncture
ME- Meningoencephalitis
CMML- Chronic Myelomonocytic Leukemia
Mo- Months
Y- Years
NR- Not Reported
ARI- Acute Renal Insufficiency
MOF-Multiple Organ Failure
DX-Diagnosis

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