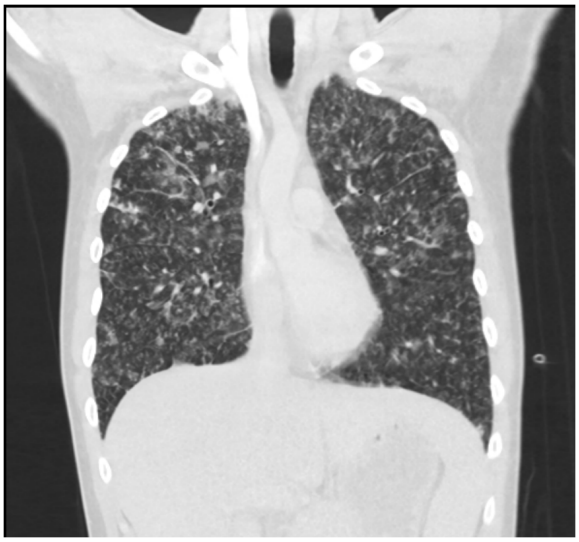
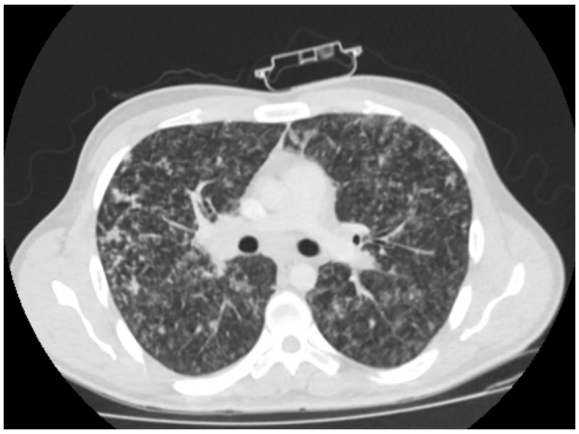




Disclosures. **All authors:** No reported disclosures.

282. Epidemiology of Candidemia in Patients with Solid Tumors of the Gastrointestinal Tract

Olga Vasylyeva, MD[1]; Tara Chen, MD[1]; John J. Hanna, MD[2]; Hamza Hassan, MD, MBBS[3] and Yetunde Ogunsesan, MBBS/MD[3]; [1]Rochester Regional Health, Rochester, New York, New York; [2]Rochester General hospital, Rochester, New York; [3]Rochester General Hospital, Rochester, New York

Session: 40. Fungal Diagnostics
Thursday, October 3, 2019: 12:15 PM

Background. The Gastrointestinal (GI) tract is considered as an important source of candidemia. Numerous studies indicate that the majority of patients with candidemia and cancer have an underlying solid tumor mostly of the GI tract. Widespread use of antifungal prophylaxis among patients with selected hematological malignancies resulted in a proportional redistribution of the frequency of candidemia among patients with various malignancies, but the incidence of candidemia among patients with GI solid malignancies is unknown.

Methods. A retrospective chart review of patients diagnosed with GI malignancies from 2010 to 2018 at Rochester Regional Health, Lipson Cancer Institute was conducted, and the incidence of candidemia was determined.

Results. A total of 2783 patients with GI malignancies were analyzed. Fifty-six percent were males, and a mean age was 67 years. Sites of malignancy included large intestine (n = 1269), pancreas (n = 394), any part of the mouth and associated organs (n = 282), liver and biliary system (n = 273), stomach (n = 235), esophagus (n = 135), small intestine (n = 110), and others (n = 85). Over the period of review, total mortality was 49%. Only 0.7% (n = 19) patients developed candidemia, with a total of 22 events. Nine episodes of candidemia happened prior to diagnosis of cancer, and 13 episodes developed after or at the time of diagnosis. There was no commonality in GI solid malignancy site among patients with candidemia. *C. albicans* was the most common isolate (9 episodes), followed by *C. parapsilosis* (8), *C. glabrata* (3), and *C. dubliniensis* (2). At the same time, there were 273 episodes of bacteremia in 230 patients (8%).

Conclusion. In our study candidemia among patients with GI solid-organ malignancies was very rare.

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

283. Potentially Achievable Hepatitis A Vaccination Coverage with Simultaneous Administration of Vaccines Among Young Children in the United States

Zhen Zhao, PhD; Holly Hill, MD, PhD; Laurie Elam-Evans, PhD; Michael Chen, PhD and James A. Singleton, PhD; [1]Centers for Disease Control and Prevention, Atlanta, Georgia

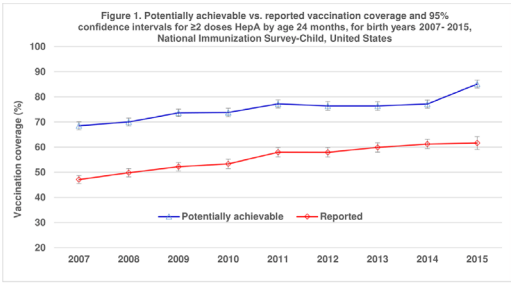
Session: 41. Hepatitis
Thursday, October 3, 2019: 12:15 PM

Background. The Advisory Committee on Immunization Practices recommends simultaneous administration of all age-appropriate doses of vaccines. We estimated the vaccination coverage for ≥2 doses of hepatitis A vaccine (≥2 HepA) that could have been achieved if opportunities for simultaneous administration with other recommended childhood vaccines had not been missed.

Methods. We analyzed National Immunization Survey-Child data for 2008–2017 in the United States. We defined potentially achievable ≥2 HepA coverage by age 24 months as the possible coverage if opportunities for simultaneous administration with other age-appropriate doses of vaccines for children by age 24 months had not been missed. We compared potentially achievable vaccination coverage to reported ≥2 HepA vaccination coverage by birth years 2007 to 2015. For children born in 2015, we stratified estimates by state and by selected socio-demographic factors. Both potentially achievable and reported ≥2 HepA coverage were evaluated using a Kaplan–Meier survival procedure to account for censoring of vaccination status.

Results. Compared with reported vaccination coverage, potentially achievable coverage for ≥2 HepA was at least 10 percentage points higher across birth years 2007 to 2015 and would have surpassed the 85% target of Healthy People 2020 for children born in 2015 (Figure 1). For the 2015 birth cohort, potentially achievable ≥2 HepA coverage exceeded the 85% Healthy People 2020 target in ten states (Figure 2). In addition, potentially achievable vaccination coverage was higher than reported coverage across all selected socio-demographic factors, with differences ranging from 20.1 percentage-points (private insurance only) to 31.7 percentage-points (non-Hispanic Black) (Table 1).

Conclusion. Potentially achievable coverage with ≥2 HepA consistently exceeded reported coverage for children from nine recent birth cohorts and across all selected socio-demographic characteristics. Coverage could increase substantially if missed opportunities were eliminated. Evidence-based interventions such as establishment of standing orders, use of provider reminders, and use of immunization information systems are recommended to increase HepA coverage among young children.



Note: The average change in vaccination coverage per annual birth year with % (95% CI): For Reported coverage: 1.92 (1.63, 2.20), P < 0.001; For Potentially achievable coverage, 1.60 (1.07, 2.13), P<0.001.

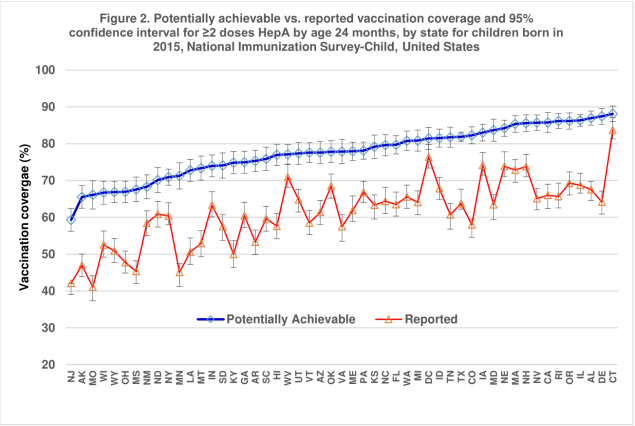


Table 1. Potentially achievable vs. reported vaccination coverage and 95% confidence intervals for ≥2 doses HepA by age 24 months, by selected socio-demographic factors, for children born in 2015, National Immunization Survey-Child, United States

Factors	Categories	Reported Coverage % (95%CI)	Potentially Achievable Coverage % (95% CI)
National		61.7(59.1-64.2)	85.1(83.5-86.6)
Children health insurance status	Private Only	68.2(64.7-71.7)	88.3(86.3-90.1)
	Any Medicaid	57.3(53.2-61.4)	83.3(80.6-85.9)
	Other Insurance	56.9(46.0-68.4)	83.1(73.7-90.5)
	Uninsured	32.5(21.5-47.2)	61.5(48.3-74.9)
Children race/ethnicity	Non-Hispanic White	61.9(58.6-65.1)	84.0(81.7-86.1)
	Non-Hispanic Black	46.8(39.9-54.2)	78.5(72.5-84.1)
	Hispanic	64.6(58.7-70.3)	87.5(84.1-90.5)
	Other	67.6(60.9-74.0)	90.0(86.3-93.0)
Family poverty level	At/Above Poverty	63.9(61.0-66.9)	87.3(85.6-88.9)
	Below Poverty	56.6(51.2-62.1)	81.1(77.2-84.6)
Residence in a metropolitan statistical area	MSA, Principal City	64.3(60.5-68.0)	86.8(84.6-88.9)
	MSA, Non-Principal City	60.3(56.2-64.5)	83.7(80.9-86.3)
	Non-MSA	56.2(50.8-61.7)	83.1(78.8-87.0)
Mother's education level	<=12 years	55.3(51.1-59.7)	81.3(78.0-84.3)
	>=13 years	65.3(62.2-68.4)	87.3(85.5-89.0)
Mother's marital status	Married	65.5(62.4-68.5)	86.8(85.0-88.5)
	Not married	53.6(47.8-59.6)	80.7(76.4-84.6)
Mother's age	Age <= 29 years	57.4(53.1-61.8)	83.7(80.7-86.5)
	>= 30 years	64.2(61.1-67.4)	85.9(83.9-87.7)
Family's mobility since birth from different state	Moved	57.3(49.8-65.1)	79.6(73.5-85.0)
	Not moved	62.2(59.5-65.0)	85.9(84.2-87.4)
Child's birth order status	Not First Born	59.0(55.7-62.3)	84.0(81.9-86.1)
	First Born	65.7(61.7-69.7)	86.6(84.1-88.9)
Vaccination provider type	Public	54.1(47.4-61.0)	81.7(76.8-86.0)
	Other Type	61.6(57.2-65.9)	85.8(83.0-88.3)
	Private	64.9(61.3-68.4)	86.4(84.2-88.5)
Number of vaccination providers for child	1 provider	63.5(60.5-66.5)	85.6(83.7-87.4)
	>=2 providers	59.2(54.4-64.1)	85.0(81.9-87.9)
Number of children in household	1 child	67.8(62.9-72.6)	88.3(85.5-90.7)
	>=2 child	59.0(56.1-62.0)	83.7(81.7-85.7)

Note: All comparisons between potentially achievable vs. reported vaccination coverage are significant at P < 0.0001.

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

284. Using Epidemiologic Investigation and Viral Sequencing to Describe and Provide Public Health Response to an Outbreak (OB) of Acute Hepatitis A Virus Infection (HAV) in the San Fernando Valley (SFV), California

Rachel H. Civen, MD[1]; Meridith Haddix, MPH[2]; Shiarron Baker, PHN[2] and Prabhu Gounder, MD[2]; [1]Community Health Services, Glendale, California; [2]Los Angeles County DPH, Los Angeles, California

Session: 41. Hepatitis

Thursday, October 3, 2019: 12:15 PM

Background. California (CA) experienced a large hepatitis A OB in 2017–2018 associated with genotype IB strains, primarily among persons experiencing homelessness and/or using drugs. In October and November 2018, we identified a cluster of three HAV cases among persons linked by drug use and homelessness in the San Fernando Valley (SFV), CA. We describe how molecular epidemiologic methods linked an additional four OB cases that lived or were associated with a senior housing facility (SHF) and guided hepatitis A vaccine outreach.

Methods. Suspect HAV cases were reported to DPH through provider and electronic lab reports with positive serum HAV IgM and resided in a 2 mile^2 area in SFV. A case report and extended interview were completed on suspects to assess risk factors associated with HAV transmission and contacts. HAV IgM positive serum specimens were sent to the CA DPH Viral and Rickettsial Disease Laboratory for HAV RNA detection and molecular sequencing. Extracted nucleic acids were amplified using nested, RT-PCR targeting the VP1-P2B region, and a 315 nt fragment was sequenced using Sanger sequencing. Contacts to cases received HAV prophylaxis and HAV vaccine outreaches occurred in at-risk settings.

Results. We identified 7 HAV cases with symptom onsets from October 2018 to January 2019. All 7 cases had positive serum HAV IgM, ≥ALT 3 X normal or had a specimen matching the OB strain and were epi- linked to a case previously identified. Of 3 homeless cases, 2 had genotype 1B, CA cluster A; one specimen was unavailable. Four additional SHF cases were 2 residents, one staff, and one visitor. Among the 4 cases associated with the SHF, three had genotype 1B, CA cluster A; one specimen was unavailable. Two elderly residents reported severe fatigue, without nausea, diarrhea and vomiting. Among the 3 homeless individuals, no direct link to the SHF was established. In total, 948 HAV vaccines were provided at the SHF, homeless shelters and other settings. HAV vaccine coverage for SHF residents and food handlers was 70% and 62%,, respectively.

Conclusion. Two clusters of HAV cases were identified among homeless persons and individuals associated with an SHF were linked through a common HAV genotype. Two elderly cases had atypical symptoms that may not have been confirmed as HAV without viral sequencing and prompted vaccine campaign to prevent additional HAV cases.

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

285. Fibrosis Progression and Clinical Outcomes in HCV/HBV Coinfected Persons in the ERCHIVES Cohort

Adeel A. Butt, MD, MS[1]; Peng Yan, MS[2]; Samia Aslam, BS[3]; Kenneth Sherman, MD, PhD[4]; Dawd Siraj, MD[5]; Nasia Safdar, MD, PhD[6] and Bilal Hameed, MD[7]; [1]Weill Cornell Medical College, Doha, Ad Dawhah, Qatar; [2]VAPHS, Pittsburgh, Pennsylvania; [3]VA Pittsburgh Healthcare System, Pittsburgh, Pennsylvania; [4]The University of Cincinnati College of Medicine, Cincinnati, Ohio; [5]University of Wisconsin, Madison, Wisconsin; [6]University of Wisconsin-Madison School of Medicine and Public Health, Madison, Wisconsin; [7]University of California at San Francisco, San Francisco, California

Session: 41. Hepatitis

Thursday, October 3, 2019: 12:15 PM

Background. Progression of liver disease and clinical outcomes in HCV/HBV coinfected persons and how they differ from HCV monoinfected persons and HCV infected persons with resolved HBV infection are not well characterized. We compared incidence of cirrhosis, hepatic decompensation and overall mortality in these three groups.

Methods. Using the Electronically Retrieved Cohort of HCV-infected Veterans (ERCHIVES), we identified those with HCV infection only, HCV/HBV coinfection (HbsAg or HBV DNA or both positive) or HCV with resolved HBV (HbcAb+ in absence of HbsAg or HBV DNA positivity). We excluded those with HIV coinfection or hepatocellular carcinoma at or before baseline, and those who received any HCV or HBV treatment. Incident rates (95% CI) were determined for cirrhosis, first hepatic decompensation event and overall mortality in the three groups.

Results. We identified 60,368 HCV monoinfected (Gp A), 151 HCV/HBV coinfected (Gp B) and 19,802 HCV infected with resolved HBV infection (Gp C). Mean age was 61.0, 60.9, and 63.0 years in the three groups and 96.5%, 96.0%, and 97.9% were males. Median baseline FIB-4 index was 2.0, 2.2, and 2.1, respectively. Incident cirrhosis (among those without cirrhosis at baseline) was increased 2- to 2.5-fold in HCV/HBV coinfected persons with baseline FIB-4 of 1.46–3.25. Hepatic decompensation and mortality were also increased several-fold in the HCV/HBV coinfected who had minimal or mild/moderate fibrosis at baseline. However, among those with cirrhosis at baseline, the difference was small among HCV/HBV coinfected and the other groups.

Conclusion. HCV/HBV coinfected persons with minimal or mild/moderate fibrosis at baseline have a much higher risk of developing cirrhosis, hepatic decompensation and mortality. However, once cirrhosis has is established, the difference is diminished. This underscores the need to intervene early when HCV/HBV coinfected persons still have minimal or mild/moderate fibrosis.

Table. Incidence rates (per 1,000 patient years of follow-up) for cirrhosis, hepatic decompensation and overall mortality.

	HCV monoinfection	HCV/HBV coinfection	HCV with resolved HBV
Cirrhosis[1]			
Baseline FIB-4 <1.45	3.55 [2.76,4.35]	0[2]	4.19 [2.47,5.9]
Baseline FIB-4 1.46-3.25	18.9 [17.6,20.2]	49.3 [6.08,92.4]	20.3 [18,22.6]
Hepatic decompensation[3]			
Baseline FIB-4 <1.45	1.71 [1.16,2.26]	0[2]	2.55 [1.22,3.89]
Baseline FIB-4 1.46-3.25	6.81 [6.03,7.59]	29.5 [-3.9,62.8]	6.57 [5.26,7.89]
Baseline FIB-4 >3.25	79.2 [75.4,83]	66.5 [8.21,125]	72.1 [66,78.2]
Mortality[3]			
Baseline FIB-4 <1.45	14.8 [13.2,16.5]	23 [-22,68.2]	19.6 [15.9,23.3]
Baseline FIB-4 1.46-3.25	16.6 [15.4,17.8]	38.3 [0.77,75.8]	16.2 [14.2,18.3]
Baseline FIB-4 >3.25	38.7 [36.2,41.2]	49.5 [0.99,98.1]	39.1 [34.8,43.4]

1 Among those with baseline FIB-4<3.25
2 There were no events in this group
3 Among all subjects

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