

while others will have intermittent or persistent CMV detection. Prior analyses assessing the association of CMV infection on hospitalization in the post-transplant period have been limited by methods that did not consider the time-varying nature of the exposure (CMV reactivation) and its confounders or aim to obtain causal effect estimates. We aimed to assess the causal effect of CMV reactivation on hospitalization using a causal modeling approach.

The Effect of CMV Infection on Hospitalization Using Generalized Estimating Equations and Marginal Structural Models

Model	Incidence Rate Ratio	95% Confidence Interval	p-value
0. GEE (TC CMV + TC Confounders)	1.10	1.04 -- 1.17	0.0014
1. GEE (TD CMV + TD Confounders)	1.22	1.12 -- 1.34	<0.001
2. MSM (TD CMV + TD Confounders)	1.03	0.91 -- 1.16	0.61

0. Generalized estimating equations treating CMV as a time-constant variable (ever present vs. not) and adjusting for baseline time-constant confounders (gender, age, race, graph versus host disease, reason for transplant)

1. Generalized estimating equations treating CMV as a time-dependent variable and adjusting for baseline and time-dependent confounders (gender, age, race, graph versus host disease, reason for transplant, history of CMV infection, history of hospitalization)

2. Marginal Structural models treating CMV as a time-dependent variable and adjusting for baseline covariates and time-dependent confounders (gender, age, race, graph versus host disease, reason for transplant, history of CMV infection, history of hospitalization)

Methods: A cohort of allogeneic HCT patients transplanted at Children's Hospital of Philadelphia January 2004–April 2017 was assembled and followed for 100 days after transplant. Eligible patients included those under CMV surveillance, defined as having ≥ 2 CMV whole blood polymerase chain reaction tests in the first month after HCT. All information was abstracted from medical charts. The association of CMV reactivation on the rate of hospitalization was estimated using traditional generalized estimating equations and repeated using a marginal structural model that accounted for time-varying exposure, confounders and non-random drop-out and obtained effects with causal interpretations.

Results: The study cohort included 340 pediatric allogeneic HCT recipients under CMV surveillance testing. 46.5% were female and the median age was 9 (range: 0 to 26). The CMV infection rate was 33.9%, with a median time to CMV detection of 23.5 days (range: 4-100). CMV infection was common in Donor+/Recipient+ (58.9%) and Donor-/Recipient+ (34.6%) patients. A traditional model estimates an additional week of CMV infection was associated with a 22% increase in average weekly hospitalization (Incidence rate ratio: 1.22, 95%: 1.12 -1.34). A marginal structure model estimates an additional week of CMV infection is associated with 3% increase in average weekly hospitalization incidence (Incidence rate ratio: 1.03, 95%: 0.91-1.16).

Conclusion: Our research showed the effect of CMV on hospitalization diminished after properly considering the time-varying nature of the CMV infection status and its confounders.

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689. Nasal versus Nasopharyngeal Sample Collection for Diagnostic Nucleic Acid Amplification Testing for Influenza A, Influenza B, and Respiratory Syncytial Viruses

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Session: P-27. Diagnostics: Virology

Background: Nucleic acid amplification testing (NAAT) for influenza A virus (IAV), influenza B virus (IBV), and respiratory syncytial virus (RSV) is a standard component of diagnosis of infections with these pathogens. At our institution, current standard of practice is to collect nasopharyngeal (NP) samples for such NAAT. In an effort to provide clinicians and patients a simpler, more comfortable sample collection option, we evaluated the use of nasal samples for NAAT, compared to NP samples.

Methods: Both nasal and NP specimens were collected from each of 58 patients seen in our emergency department (January – March 2020). NP samples were collected using minitip FLOQswabs; nasal samples were collected using regular or minitip FLOQswabs. Nasal and NP samples were processed using the same protocol and tested for influenza viruses and RSV using the Cepheid GeneXpert (Xpress Flu/RSV) platform.

Results: In total, 20 NP samples tested negative for virus and 38 tested positive (16 IAV-positive, 14 IBV-positive, 8 RSV-positive). There were 3 cases (5% of total cases) in which qualitative (positive/negative) results from the corresponding nasal samples were not in agreement with results derived from NP samples. These were considered false-negative results; one such discrepancy was resolved upon re-testing the same samples. Overall positive percent agreement between nasal- and NP-derived results was 92% (35/38), and negative percent agreement was 100% (20/20). Among samples testing positive for virus by both NP and nasal sampling methods, we found that the average cycle threshold (Ct) value for IAV detection was 5.1 cycles (n = 16, SEM = 0.83) higher for nasal samples than for NP samples. The average Ct for IBV detection was 3.3 cycles (n = 12, SEM = 1.87) higher for nasal than for NP samples. The average Ct for RSV detection was 1.9 cycles (n = 7, SEM = 1.57) higher for nasal than for NP samples.

Conclusion: These results suggest that recovery of viral RNA from nasal samples is lower than that from nasopharyngeal samples, particularly for influenza viruses. This

decreased detection of viral RNA from nasal samples may explain the false-negative results seen in our discrepant cases. These data suggest that a decrease in recovery of viral RNA by nasal sampling may translate to decreased diagnostic accuracy.

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690. Oka-Strain Varicella-Zoster Virus Meningitis in a Healthy Adolescent

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Background: Routine vaccination with varicella-zoster (VZV) vaccine has resulted in significant declines in the incidence of VZV cases, hospitalizations, and deaths across pediatric age. This vaccine is safe and effective and adverse events are monitored closely.

Methods: We describe a case of vOka meningoencephalitis in a twelve-year-old vaccinated boy who presented with altered mental status and a vesicular facial rash.

Results: A twelve-year-old healthy, fully-vaccinated boy presented to urgent care clinic with left-sided frontotemporal headache, left-sided eye pain, and photosensitivity. Over several days, a left-sided facial rash progressed to include papular and vesicular lesions over the cheek, as well as over the left side of the chin and at the midline of the lower lip. He was somnolent, sleeping 18-20 hours a day. The child was evaluated by a pediatric neurologist who noted a left-sided ptosis and left lateral rectus palsy; he was admitted for further workup. Cerebrospinal fluid (CSF) analysis showed WBC of 33 cells/ml³ with 92% lymphocytes; glucose of 44mg/dL (serum glucose 84mg/dL), and protein of 50mg/dL (range: 15-45). Nasopharyngeal multiplex polymerase chain reaction (PCR) (BioFire Diagnostics, Salt Lake City, Utah) was positive for rhinovirus/enterovirus. Testing of facial vesicles for varicella-zoster virus (VZV) and herpes simplex virus (HSV) was negative by DFA and culture, and enteroviral throat and rectal PCRs were negative. However, CSF PCR for VZV was found to be positive. In light of this finding, the viral isolate was sent to Dr. Anne Gershon's research lab at Columbia University Medical Center for typing and was determined to be vOka. Quantitative and functional immune studies were performed, and were normal. The patient initially received 7 days of intravenous acyclovir, during which time his rash resolved and mental status returned to baseline. He completed a total of 14 days of acyclovir and has had no recurrences.

Conclusion: This case represents only the tenth case of Oka-strain meningitis in an immunocompetent child reported to date, and one of very few cases in immunocompetent adolescents. While rare, vOka meningitis is an entity of which primary care pediatricians and infectious diseases specialists should be aware, even in older children.

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691. Results of Repeat HIV-1 DNA Resistance Tests Are Highly Concordant

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Background: DHHS guidelines recommend caution when interpreting HIV-1 DNA resistance testing because not all previously identified drug resistance mutations (DRMs) may be captured. Comparison of multiple reports from the same patient was performed to assess the ability of HIV-1 DNA testing to consistently identify wild-type and drug resistance alleles.

Methods: Patients with 3 HIV-1 DNA resistance tests (trios) and corresponding HIV-1 viral load (VL) measurements within ~3 months of each resistance test were identified in a commercial database. Concordance among trio test results was assessed for each patient. VL and trio timespan were evaluated for impact on concordance using one-way ANOVA with post-hoc analyses.

Results: Fifty-five patients with test trios were identified for analysis. Average patient age was 53 years, and 88% were male. All 3 tests within 26/55 trios were associated with VL < 200 copies/mL. The average testing timespan was 100 weeks (range 17-228 weeks). Wild-type virus was identified on 3/3 reports for 11/55 (20%) patients. Among resistant viruses, DRMs were identified on average 70%, 20% and 10% on 3/3, 2/3 and 1/3 of HIV-1 DNA reports, respectively. M184V was identified on 3/3, 2/3 and 1/3 reports among 14/17, 2/17, and 1/17 test trios, respectively. K103N was identified on 3/3 and 2/3 reports among 11/12 and 1/12 test trios, respectively. The redetection rate following an initial HIV-1 DNA test was high for M184V (30/34, 88%) and K103N (23/24, 96%).

Of 178 DRMs detected across all initial HIV-1 DNA tests, 17 (9.6%) were not detected on the second, but redetected on the third test, including M184V and K103N in one trio each. The average concordance among test trios across all drug classes was 97%. No correlation between VL at time of testing and DRM redetection rates was observed. Significantly fewer DRMs were recaptured when repeat testing was performed > 24 months after the initial test.

Conclusion: Repeat HIV-1 DNA drug resistance testing reliably detected archived DRMs. DRM decay related to turnover of the viral reservoir may explain some discordance between repeat HIV-1 DNA tests.

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