Disclosures. All authors: No reported disclosures.


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Background. GCV-R CMV infection is an emerging cause of morbidity and mortality in lung transplant recipients. The purpose of this study was to evaluate the clinical and economic impact of GCV-R CMV infection in a high-risk population.

Methods. We performed a single-center, retrospective cohort study of lung transplant recipients with genotype confirmed GCV-R CMV and ganciclovir-sensitive (GCV-S) CMV infection, matched (1:3) by year of diagnosis. Clinical outcomes within 1 year following the onset of CMV infection and total hospital costs were assessed.

Results. Twenty-eight patients were included in the analysis: 7 with GCV-R CMV infection and 21 with GCV-S CMV infection. Baseline demographics (Table 1) were similar in the two groups. CMV load at diagnosis was numerically higher (282,932 vs. 34,574 IU/mL, P = 0.01) and days to CMV infection following discontinuation of antiviral prophylaxis was numerically lower (20 vs. 150 days, P = 0.07) in the GCV-R CMV group. All-cause mortality (71.4% vs. 19.0%, P = 0.02) and total hospital days due to CMV infection (63 vs. 34-76 days, P = 2.9, P = 0.01) were significantly higher in the GCV-R CMV cohort. There were no differences in allograft rejection and hospital readmission between the two groups. Total hospital costs were significantly higher amongst patients with GCV-R CMV infection ($208,924 vs. $20,419, P = 0.02) and patients with GCV-S CMV infection were discharged from the hospital earlier (14 vs. 71 days, P = 0.01).

Conclusions. GCV-R CMV infection is associated with poor outcomes and considerable healthcare costs. Novel prophylaxis and treatment strategies are needed to combat CMV infection in lung transplant recipients.

Table 1. Baseline Demographics

<table>
<thead>
<tr>
<th></th>
<th>GCV-R CMV [N = 7]</th>
<th>GCV-S CMV [N = 21]</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at transplant, years</td>
<td>57 (56-61)</td>
<td>46 (29-61)</td>
<td>0.189</td>
</tr>
<tr>
<td>Male</td>
<td>6 (85.7)</td>
<td>13 (61.9)</td>
<td>0.371</td>
</tr>
<tr>
<td>Indication for transplant</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cystic fibrosis</td>
<td>1 (14.3)</td>
<td>8 (38.1)</td>
<td>0.371</td>
</tr>
<tr>
<td>Idiopathic pulmonary fibrosis</td>
<td>3 (42.9)</td>
<td>5 (23.8)</td>
<td>0.371</td>
</tr>
<tr>
<td>COPD</td>
<td>1 (14.3)</td>
<td>5 (23.8)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>1 (14.3)</td>
<td>1 (4.8)</td>
<td>0.444</td>
</tr>
<tr>
<td>Other</td>
<td>1 (14.3)</td>
<td>2 (9.5)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Type of transplant</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bilateral</td>
<td>5 (71.4)</td>
<td>18 (85.7)</td>
<td>0.574</td>
</tr>
<tr>
<td>Single</td>
<td>2 (28.6)</td>
<td>3 (14.3)</td>
<td>0.574</td>
</tr>
<tr>
<td>CMV serostatus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D+/R-</td>
<td>6 (85.7)</td>
<td>18 (85.7)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>D+/R+</td>
<td>1 (14.3)</td>
<td>3 (14.3)</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Duration of CMV prophylaxis after transplant (days)</td>
<td>175.5 ± 54.3</td>
<td>164.9 ± 63.5</td>
<td>0.669</td>
</tr>
</tbody>
</table>

Disclosures. T. Patel, Merck: Grant Investigator, Research grant. K. Gregg, Merck: Grant Investigator, Research grant.

2443. BK Polyoma Virus Nephropathy in Hematopoietic Cell Transplant Recipients with Renal Dysfunction

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Background. BK polyoma virus (BKV) nephropathy (BKVN) is a well-established cause of allograft loss after kidney transplantation. In contrast BKVN is rarely been reported in hematopoietic cell transplant (HCT) recipients. Renal dysfunction after HCT is common and often attributed to total body irradiation, drug toxicity, hypertension or microangiopathy. As kidney biopsies are rarely performed after HCT, BKVN may be underdiagnosed. We report a single-center experience of BKVN in HCT recipients.

Methods. Retrospective chart review of HCT recipients from January 1, 2016 through March 31, 2017. Only cases of BKVN confirmed by immunohistochemical stain on renal biopsy are included. Urine and blood BKV PCR was performed at Vircor Eurofins (Lee’s Summit, MO). Glomerular filtration rate (GFR) was estimated by Chronic Kidney Disease Epidemiology Collaboration equation.

Results. From 2016 to 2017, 320 patients received HCT and 6 patients underwent kidney allograft transplantation for BKV infection. Patient characteristics are shown in Table 1. Three patients (75%) received ex vivo T-cell depleted (CD34+ selected) peripheral blood (PB) HCT and did not receive pharmacologic GVHD prophylaxis; one patient received cord blood allograft. All patients had BKV viremia with a median BKV viral load of 9.3 log10 copies/mL (range, 8.6-10.0) and median onset 18 days (range 6-41) post HCT. BKVN was diagnosed at a median of 275.5 days post-HCT (range, 141-637). The patients presented with decreased GFR (median 47.5% reduction, range 16-75%) from GFR at transplant. One patient had proteinuria (3 g over 24 hours); one patient had hydronephrosis. At BKVN diagnosis plasma BKV viral load was a median of 6.2 log10 copies/mL, range 6.0-6.3, absolute lymphocyte count median 1027 (range 335-2536) and CD4+ lymphocyte count median 145 (range 64-172).

Conclusion. BKVN should be considered in HCT recipients with worsening renal function and high BKV viremia. Early, noninvasive predictors of BKVN could aid in identifying high-risk patients for early intervention prior to irreversible loss of kidney function. (3) Reduction of immunosuppression is often not feasible in HCT. The role of preemptive antiviral therapy and/or adoptive cell therapy for BKV viremia in HCT should be evaluated in clinical trials.

Disclosures. G. Papasoulioua, Chimerix: Consultant, Grant Investigator and Investigator, Consulting fee, Grant recipient and Research grant.

2444. Coronavirus Infection in Hematopoietic Stem Cell Transplant Recipients

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Background. Hematopoietic stem cell transplants (HSCT) recipients are at increased risk of respiratory viral infections and their associated complications. Although the epidemiology of many respiratory viruses has been well characterized in this population, little is known about the epidemiology of human coronavirus (HoCV) infection.

Methods. We identified HSCT recipients with symptoms of a respiratory tract infection who tested positive for HoCV by nasopharyngeal (NP) swab from January 2013 to December 2016 at our hospital. NP swabs were analyzed by the FilmArray® Respiratory Panel, which detects 17 respiratory viruses, including 4 coronavirus serotypes. We reviewed the demographics, transplant type, comorbidities, smoking status, respiratory symptoms, co-pathogens, and radiographic findings of infected patients. We then assessed the incidence of developing a lower respiratory tract infection (LRTI), defined as new pulmonary infiltrates or detection of HoCV in bronchoalveolar lavage fluid, within 30 days of initial diagnosis.

Results. We identified 58 HSCT recipients who tested positive for HoCV. The median patient age was 54 years, 29 (50%) were men, and 24 (41%) were current or prior smokers. Fifty (86%) patients had received an allogeneic HSCT and 8 (14%) had
received an autologous HSCT. The coronavirus serotypes were: OC43 (n = 19, 33%), NL63 (n = 18, 31%), HKU1 (n = 16, 28%), and 229E (n = 5, 9%). The median time from transplant until detection of HSCT infection was 135 days (IQR=256). Seventeen (29%) patients were lymphopenic at the time of diagnosis and 17 (29%) were receiving corticosteroids. The most common initial symptoms were cough (n = 41, 71%), rhonchi (n = 31, 53%), and dyspnea (n = 17, 28%), and 19 (38%) and 16 (28%) patients had fever and hypoxia, respectively. Seventeen patients (29%) developed a LRTI within 30 days of diagnosis and 43 harbored a co-pathogen in the blood or respiratory tract. Three patients (5%) were intubated for respiratory failure and 1 (2%) died of 63 patients during the study period.

Conclusion. HSCT infection is common in HSCT recipients and is caused by multiple serotypes. Nearly one-third of patients have fever and hypoxia upon initial diagnosis or progress to LRTI. Further research is needed to identify risk factors for HSCT LRTI in this population.

Disclosures. All authors: No reported disclosures.

2445. Respiratory Viral Infections in Multiple Myeloma Patients
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Session: 259. Viral Infections in Transplantation
Saturday, October 7, 2017: 12:30 PM

Background. Multiple myeloma (MM) patients are at increased risk of respiratory viral infections (RVIs) due to disease-related alterations in their immune systems. Data in the literature specific to MM patients is limited. We reviewed four years of multisite respiratory viral panel (RVP) data in MM patients at our institution to evaluate incidence and epidemiology of RVIs. Methods. The results from all positive RVPs obtained via nasopharyngeal swab and as identified by polymerase chain reaction during the years 2013 to 2016, were analyzed. A positive result less than 6 weeks apart was considered a duplicate and removed. All specimens were analyzed in the molecular diagnostics laboratory using the Accuplex Respiratory Viral Panel (GenMark, Dr. Carlsbad, CA). This assay is a qualitative nucleic acid multiplex in vitro diagnostic test that provides for the simultaneous detection and identification of 14 respiratory viral nucleic acids. Results. RVIs were reported in every month in all four years. The peak months were January and February, driven by the peak activity of Influenza and respiratory syncytial virus (RSV). Rhinovirus was isolated the most frequently. The least isolated was Adenovirus. A seasonality was observed with Influenza, RSV, human para influenza and human metapneumovirus; however, infections with each virus occurred outside of peak months including an outbreak of Influenza during July and August 2013. The total number of viral infections varied each year as did the total number for each virus. The year 2015 had the lowest number of RVIs reported at 427, followed by the year 2016 with the most RVIs reported at 515. However, 2016 was not the peak year of RVIs due to disease-related alterations in their immune systems. Among adults hospitalized with influenza, IC patients had worse outcomes including a longer duration of hospitalization and higher probability of pneumonia and intensive care unit admission, and increased all-cause mortality, although these results are not adjusted for potential confounders.


2446. Clinical Features and Outcomes of Immunocompromised Adults Hospitalized with Laboratory-confirmed Influenza in the USA, 2011–2015

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Session: 259. Viral Infections in Transplantation
Saturday, October 7, 2017: 12:30 PM

Background. During the 2011–2015 influenza seasons, the National Influenza Surveillance System reported to CDCs Influenza Hospitalization Surveillance Network (FluSurv-NET). We defined IC patients as having ≥2 of the following: HIV, AIDS, cancer, stem cell or organ transplantation, non-steroid immunosuppressive therapy, immunoglobulin deficiency, asplenia, and other rare conditions. We compared IC and non-IC patients using χ² or Fishers’s exact tests and t-tests or Mann–Whitney U tests.

Results. Among 35,348 adults hospitalized over four seasons, 3,633 (10%) were IC. The most common IC conditions were cancer (4%), non-steroid immunosuppressive therapy (4%), and HIV (17%). IC patients were younger than non-IC patients (mean 61 ± 17 yrs. vs. 67 ± 20 yrs; P < 0.01). IC patients were more likely to have underlying renal disease (27% vs. 18% and liver disease (7% vs. 3%) and less likely to have most other chronic underlying conditions including obesity (18% vs. 23%), cardiovascular disease (40% vs. 47%), and chronic lung disease (35% vs. 41%; P < 0.01 for all). IC patients were more likely to have received influenza vaccination (53% vs. 46%; P < 0.01). Among cases with symptom data (2014–2015), IC patients were more likely to present with fever (68% vs. 61%; P < 0.01) but respiratory distress was similar (53% vs. 54%; P = 0.3). Overall, the majority of IC and non-IC patients received antivirals (87% vs. 85%; P < 0.01). IC patients had a longer duration of hospitalization (median [IQR] 4 (2–6) vs. 3 (2–6) days; P < 0.01) and were more likely to be diagnosed with pneumonia (34 vs. 31%; P < 0.01) and to require intensive care (18% vs. 16%; P = 0.01). Death during hospitalization occurred in 135 (3.7%) IC and 945 (3.0%) non-IC patients (P < 0.01).

Conclusion. Among adults hospitalized with influenza, IC patients had worse outcomes including a longer duration of hospitalization and higher probability of pneumonia and intensive care unit admission, and increased all-cause mortality, although these results are not adjusted for potential confounders.

Disclosures. All authors: No reported disclosures.

2447. Incidence and Outcomes of Cytomegalovirus (CMV) Infection among Hematopoietic Stem Cell Transplant (HSCT) Recipients

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Session: 259. Viral Infections in Transplantation
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Background. Outcomes of CMV infection among HSCT recipients likely vary by patient population and treatment modality. However, data on these outcomes have been reported by relatively few centers.

Methods. This was a retrospective cohort study of allogenic HSCT recipients age 18 years at Oregon Health and Science University Hospital (OHSU) between 2010–2015. During the study period, OHSU standard practice was to preemptively treat CMV viremic patients (quantitative PCR assay ≥200 copies/mL or consecutive PCR assays ≥200 copies/mL) with first-line ganciclovir or ganciclovir and second line foscamet if there were contraindications to first-line agents. Study data were collected from an electronic health record repository and local Center for International Blood and Marrow Transplant Research (CIBMTR) database. Primary outcomes were clinical manifestations of CMV disease, death, and cause of death within 1 year of transplant.

Results. Among 409 HSCT recipients, mean age was 53 (standard deviation: 13) years and 41% were female. 192 (47%) patients had CMV viremia and the median (interquartile range) time to CMV reactivation was 42 (31–53) days (Figure 1). Patients with myeloid leukemia were more likely to have CD4+ T cell lymphopenia (50% vs. 29%, P < 0.06). 4 (1%) patients had a documented clinical manifestation of CMV disease (3 pneumonia and 1 pancreatitis). One-year mortality was 10% (20/202). There was no significant difference in mortality (37.5% vs. 35.0%, P = 0.60) or cause of death (P = 0.30) between patients with and without CMV reactivation (Figure 2). The most frequent causes of death among CMV viremic patients were recurrent/persistent disease (35%), acute graft vs. host disease (GVHD) (22%), infection (19%), and chronic GVHD (11%). CMV was documented as the primary cause of death for 2 patients.