copies/mL) or negative viral load. Sustained EBV DNAemia was defined as EBV DNA detection (> 200 copies/mL) on at least 3 consecutive samples. Categorical variables were analyzed using chi-square or Fishers exact test. Mann-U Whitney test was used for continuous variables.

### Results

442 SOT recipients (258 kidney, 141 liver, 22 kidney-pancreas (KP), 15 kidney-liver, 6 pancreas) were examined. Most subjects (430, 97%) were EBV intermediate-risk (recipient (R)+). 8 subjects (2%) were EBV high risk (donor (D)+, R-) and 4 (1%) were EBV low risk (D-/R-). EBV viral loads were obtained in 177/442 (40.0%) recipients. DNAemia was detected in 18/442 subjects (4.1%). It was most common in pancreas recipients (1/6; 16.7%) compared with kidney (10/258; 3.9%), liver (5/141; 3.5%), and KP recipients (1/22; 4.5%). DNAemia was most frequently observed in the D+/R+ (3/8; 37.5%) group compared to intermediate risk (R+) (15/430; 3.5%) and D-/R- (0%) groups. Median time to EBV viral load detection was 14 months (range 3-60). In univariate analysis, EBV high-risk serostatus was the factor most strongly associated with development of EBV DNAemia (P < 0.001). Sustained DNAemia (median viral load 1829 copies/mL; peak viral load 1.9 million copies/mL) was observed in 8 subjects (1.8%). PTLD developed in 6 subjects (1.4%); 50% had sustained DNAemia prior to diagnosis.

### Conclusion

While uncommon, development of sustained EBV DNAemia was associated with subsequent development of PTLD in our cohort of adult SOT recipients. These data provide guidance for identifying subjects at risk for PTLD above and beyond baseline EBV high-risk serostatus.

### Disclosures

All Authors: No reported disclosures

### 936. Risk Factors and Clinical Outcomes for Extended-Spectrum Beta-Lactamase Producing Enterobacterales Blood Stream Infections in Patients with Hematologic Malignancies

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### Background

Hematologic malignancy patients have high rates of antibiotic exposure, and increasing resistance is a major concern, particularly with extended-spectrum beta-lactamases (ESBL) in Enterobacteriaceae blood stream infections (BSIs). Identifying risk factors for ESBL-producing Enterobacteriaceae (ESBL-E) BSIs may facilitate faster appropriate antibiotic use and decrease mortality.

### Methods

This was a retrospective study of patients with hematologic malignancies with Enterobacteriaceae bacteremia admitted to Carolinas Medical Center in Charlotte, NC from January 2010 through September 2020. The primary objective was to compare 30-day mortality rates for patients with ESBL-E BSIs to those with non-ESBL-E BSIs. Fisher’s exact or Mann-Whitney U tests were used for primary and secondary clinical outcomes as appropriate. Risk factors associated with 30-day mortality and ESBL BSI production were assessed as secondary objectives using logistic regression models.

### Results

A total of 28 patients with ESBL-E BSIs and 60 patients with non-ESBL-E BSIs were included. The 30-day mortality rate with ESBL-E BSIs was 25% compared to 15% with non-ESBL-E BSIs (P = .373). In-hospital mortality, 30-day infection recurrence, intensive care unit (ICU) admission, and length of stay after culture were not significantly different. However, time to optimal therapy was longer in the ESBL-E group (median 42.3 vs 1.9 hr; P = .001). Multivariate logistic regression analysis showed an association of 30-day mortality with ICU admission (OR 16.7; 95% CI, 3.56-78.4; P < .001) and later to optimal therapy (OR 1.03; 95% CI, 1.01-1.05; P = .026). Prior ESBL-positive culture was associated with ESBL-E BSIs on univariate logistic regression (OR 9.83; 95% CI, 1.05-92.3; P = .049). Additionally, prolonged neutropenia (OR 3.05; 95% CI, 1.01-9.23; P = .049) and prior invasive antibiotic use (OR 2.96; 95% CI, 0.96-9.09; P = .059) were associated with ESBL-E BSIs in the multivariate analysis.

### Conclusion

Significantly longer time to optimal therapy was seen in ESBL-E BSIs and was associated with mortality in patients with hematologic malignancies. The identified ESBL risk factors create an opportunity to decrease delay in optimal therapy through risk stratification during initial antibiotic selection.

### Disclosures

Ekaterina Kachur, PharmD, BCOP, Bristol Myers Squibb (Advisor or Review Panel member); Kyowa Kirin (Advisor or Review Panel member); Genterch (Employee/Glassomikihlke (Advisor or Review Panel member)