1124. Effects of Co-infection on the Severity, Response to Treatment and Duration of Hospital Stay in Patients with Clostridium difficile Infection
Muhammad Shafig, MD1; Hani Alturkman, MD2; Yousaf Zafar, MD2; Vishal Mittal, MD1; Hafsa Lodhi, MD1; Joseph Brewer, MD2; 1Internal Medicine, University of Missouri, Kansas City – School of Medicine, Kansas City, Missouri, 2Medical Student, University of Missouri, Kansas City – School of Medicine, Kansas City, Missouri, 3St. Luke’s Hospital, Kansas City, Missouri
Session: 133. Enteric Infections
Friday, October 5, 2018: 12:30 PM
Background. According to the multicenter evaluation of the FilmArray® multiplex gastrointestinal (GI) panel for etiologic diagnosis of infectious gastroenteritis, the GI panel detected at least one potential pathogen in 53.5% of the stool specimens that were collected. Out of the positive samples, 31.5% tested positive for more than one potential pathogen. The samples that were co-infected showed that Closstridium difficile infection (CDI) was present in 53.4% of them. This lead to the idea of our project to determine whether the presence of another GI infection affects CDI outcomes in terms of severity, treatment escalation, duration of hospital stay and recurrence.
Methods. Inclusion criteria: 18-year-old and above patients. Exclusion criteria are GI panel performed on outpatient basis, presence of any co-founder that had independent effect on the outcomes such as end-stage renal disease, cirrhosis, presence of non-GI infection (pneumonia, urinary tract infection, osteomyelitis etc.), and recurrent CDI. Out of the 2,576 GI panels performed from January 1, 2015 until December 31, 2016 only 235 patients were selected for retrospective chart review based on the above criteria.
Results. Out of 235 patients, 38 patients had co-infection (GI + another GI infection = Group A) and reminder had only GI (Group B). Chi-square test, Fisher’s exact test (for severity, treatment escalation and recurrence) and Independent T-test (for duration of hospital stay) were used to compare Group A with Group B. Alpha criterion was 0.05.
Conclusion. All the resulted P-values are greater than 0.05. These results are suggestive of the fact that presence of another GI infection does not affect the outcomes for CDI in terms of treatment escalation, duration of hospital stay and recurrence. As there were only 38 patients in co-infection group, it limits the ability to determine the effect of individual infectious agent on the outcomes of CDI.
Disclosures. All authors: No reported disclosures.

1125. Reduced Time to Pathogen Identification and Antibiotic Prescription Using Multiplex Molecular Testing for Gastrointestinal Infections
Aleksandra Kardasheva, DO1; Evan Yount, MD1; Tracie Rose, Technical specialist2; Purnam Verma, PhD1 and Shingo Chihara, MD1, 1Graduate Medical Education, Virginia Mason Medical Center, Seattle, Washington, 2Microbiology, Virginia Mason Medical Center, Seattle, Washington, 3Internal Medicine, Section of Infectious Diseases, Virginia Mason Hospital and Seattle Medical Center, Seattle, Washington
Session: 133. Enteric Infections
Friday, October 5, 2018: 12:30 PM
Background. A variety of microbial pathogens causes diarrhea which remains a significant global concern. The ability to rapidly identify the pathogen impacts the decision to treat, promotes antimicrobial stewardship, and assists with infection control and prevention. The objective of the study was to compare the time it took for rapid identification of microbial pathogens via a stool-culture based testing vs. real-time PCR using a Verigene Enteric Pathogens (EP) test.
Results. In 2015, 2,194 stool specimens were tested and 136 (6.2%) were positive. The median time to prescription in 2015 was 53.84 hours in comparison to 21.96 hours in 2017. Years 2015 and 2017 were chosen as in 2016 the culture-dependent testing was started on chemo and day 8 at which time all patients were neutropenic. The patients developed fevers, abdominal pain, and diarrhea within 36 hours of starting midostaurin and had abdominal CT findings consistent with neutropenic enterocolitis. For two patients, midostaurin was discontinued and symptoms improved upon discontinuation. One patient completed the course of midostaurin with symptom resolution after its completion. Of note, all were started on appropriate prophylactic antibiotics at chemotherapy initiation and were started on broad-spectrum antibiotics at onset of fever and abdominal symptoms. Appropriate evaluation was also done for each patient to rule out other causes of abdominal pain. A majority of patients were neutropenic who began reporting symptoms of neutropenic enterocolitis within 36 hours of receiving midostaurin. This shows a possible increased toxicity when midostaurin is given after induction chemotherapy in the setting of neutropenia. Stone et al. showed increased intestinal symptoms with midostaurin, but no cases of neutropenic enterocolitis have been reported. With increased midostaurin use in the past year, further studies are warranted to establish and raise awareness of a possible direct association between midostaurin and gastrointestinal toxicity.
Disclosures. All authors: No reported disclosures.

1126. Three Cases of Neutropenic Enterocolitis Following Midostaurin Administration
Ryan Carroll, MD1; Courtney Nichols, MD1; Hesham Awadh, MD2; Harsh Vyasweshwar, MD1; Derek Evans, Pharm.D3 and Kara Willenburg, MD4; 1Internal Medicine, Marshall Joan C. Edwards School of Medicine, Huntington, West Virginia, 2Department of Medicine and Pediatrics, Marshall Joan C. Edwards School of Medicine, Huntington, West Virginia, 3Internal Medicine, Marshall University – Joan C. Edwards School of Medicine, Huntington, West Virginia, 4Internal Medicine, Marshall University – Joan C. Edwards School of Medicine, Huntington, West Virginia
Session: 133. Enteric Infections
Friday, October 5, 2018: 12:30 PM
Background. Neutropenic enterocolitis is a life-threatening inflammation of the colon with a mortality rate above 50% primarily seen in neutropenic patients on cytotoxic chemotherapy. The following cases illustrate three patients with this condition following midostaurin administration after standard induction chemotherapy with daunorubicin/idarubicin and cytarabine for acute myeloid leukemia (AML). Midostaurin is a multiaffected FMS-like tyrosine kinase 3 (FLT3) receptor inhibitor used in AML treatment after induction chemotherapy.
Methods. Review of records of three patients seen by the infectious disease service.
Results. In these cases, patients were diagnosed with AML with FLT3 mutation. All three were admitted and started on standard induction chemotherapy. Midostaurin was started on chemo and day 8 at which time all patients were neutropenic. The patients developed fevers, abdominal pain, and diarrhea within 36 hours of starting midostaurin and had abdominal CT findings consistent with neutropenic enterocolitis. For two patients, midostaurin was discontinued and symptoms improved upon discontinuation. One patient completed the course of midostaurin with symptom resolution after its completion. Of note, all were started on appropriate prophylactic antibiotics at chemotherapy initiation and were started on broad-spectrum antibiotics at onset of fever and abdominal symptoms. Appropriate evaluation was also done for each patient to rule out other causes of abdominal pain. A majority of patients were neutropenic who began reporting symptoms of neutropenic enterocolitis within 36 hours of receiving midostaurin. This shows a possible increased toxicity when midostaurin is given after induction chemotherapy in the setting of neutropenia. Stone et al. showed increased intestinal symptoms with midostaurin, but no cases of neutropenic enterocolitis have been reported. With increased midostaurin use in the past year, further studies are warranted to establish and raise awareness of a possible direct association between midostaurin and gastrointestinal toxicity.
Disclosures. All authors: No reported disclosures.

1127. Genomic Analysis of Biofilm-Forming Enteroinvasive E. coli Emergent Pathogen
Oscar Gomez-Duarte, MD, PhD1; Julio Guerra, MS2 and Ricky Ko, Student2; 1Pediatrics, University at Buffalo, Buffalo, New York, 2University at Buffalo, Buffalo, New York
Session: 133. Enteric Infections
Friday, October 5, 2018: 12:30 PM
Background. Enteroinvasive Escherichia coli (EIEC) are involved in dysenteric diarrhea among children in low- and middle-income countries. EIEC strains isolated in Colombia, South America were shown to form biofilms and to be invasive in vitro. The O96:H1 serotypes and biofilm formation (BF) are not common phenotypes among EIEC, and the role they may play in diarrhea is at present unknown. The main goal of this study was to identify virulence and BF genes from EIEC genomic data. We hypothesize that EIEC O96:H1 strain 52.1 originated from horizontal transfer of a Shigella-like virulence plasmid into a non-EIEC pathogenic E. coli strain.
Methods. WGS was performed on the BF-EIEC 52.1 strain using NextGen Illumina and Pacific Biosciences (PacBio) platforms. Publically available genomes from other EIEC O96:H1 and Shigella genomes previously published were analyzed using online available software and databases including NCBI, BLAST, Mauve, among others. This analysis was tailored to identify virulence factors from the virulence factor database (VFDB), BLAST and to determine identity and query coverage of genes encoding the Shigella virulence factors. EIEC and Shigella genomes were analyzed on a multiple genome alignment software (Mauve) to verify results from BLAST and to determine pseudogenes.
Results. The genome of EIEC O96:H1 serotype 52.1 was 5,193,449 bp in size, containing 5,050 coding DNA sequences (CDSs). O96:H1 strain 52.1 carries three plasmids, the invasion plasmid (pINV) contains all type 3 secretion system (TTSS) and TTSS effectors genes previously described for Shigella and EIEC O96:H1 CFA/SE10297/87 Italian strain. Non-TTSS virulence genes were also identified, including long polar fimbrial gene (lpfA), enterotoxin (senB), and antibiotic resistance genes.
Conclusion. The EIEC O96:H1 strain 52.1 genome carries TTSS genes within a virulence plasmid, protein effector genes, and enterotoxin genes known to be