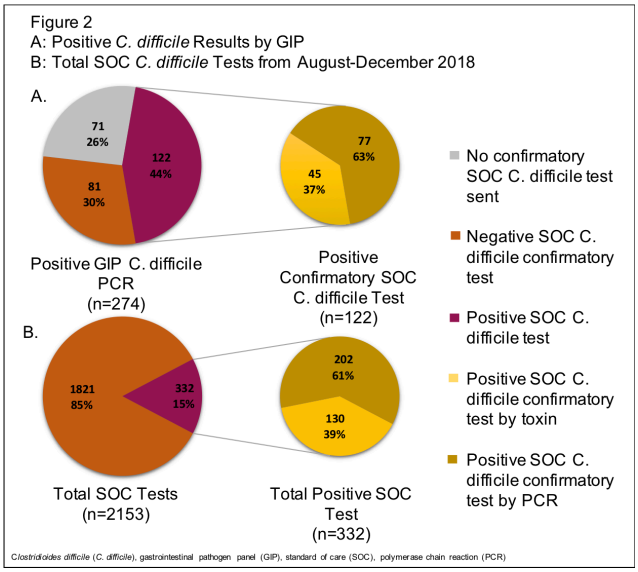

Table 2. Clinical Outcomes of Study Groups

Clinical Outcome	GIP (+) only n=81, (%)	SOC PCR (+) n=77, (%)	SOC Toxin (+) n=45, (%)	Control group n=160, (%)	Global p-value*
Mortality	3 (4)	4 (5)	3 (7)	9 (6)	0.89
30-day re-admission	13 (16)	13 (17)	35 (22)	27 (17)	0.83
True CDI by SOC test within 3 months	3 (4)	9 (12)**	7 (16)**	1 (1)	<0.001
True CDI by SOC test within 6 months***	0 (0)	4 (5)	2 (4)	5 (3)	0.26

Gastrointestinal pathogen panel (GIP), standard of care (SOC), polymerase chain reaction (PCR), positive (+), *Clostridioides difficile* infection (CDI)
*Fisher's Exact Test.
**Significantly different from control group (p-value <0.001).
***16 people with initial *C. difficile* positive GIP test <6months are not included in current analysis.

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

2355. The Association Between Diagnostic Testing Method and *Clostridium difficile* Infection Severity

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Background. The optimal diagnostic strategy for Clostridium difficile infection (CDI) is not known, and no test is shown to clearly differentiate colonization from symptomatic infection. We hypothesized that detection and/or quantification of stool toxins would associate with severe disease and adverse outcomes.

Methods. We conducted a retrospective cohort study among subjects with CDI diagnosed in 2016 at the University of Michigan. The clinical microbiology laboratory tested for glutamate dehydrogenase antigen and toxins A/B by enzyme immunoassay (EIA). Discordant results reflexed to PCR for the tcdB gene. Stool toxin levels were quantified via a modified cell cytotoxicity assay (CCA). *C. difficile* was isolated by anaerobic culture and ribotyped. Severe CDI was defined by the IDSA criteria: white blood cell count >15,000 cells/μL or a 1.5-fold increase in serum creatinine above baseline. The primary outcomes were all-cause 30-day mortality and a composite of colectomy, ICU admission, and/or death attributable to CDI within 30 days. Analysis included standard bivariable tests and adjusted models via logistic regression.

Results. From 565 adult patients, we obtained 646 samples; 199 (30.8%) contained toxins by EIA. Toxin positivity associated with IDSA severity (Table 1), but not our primary outcomes on unadjusted analysis. After adjustment for putative confounders, we still did not observe an association between toxin positivity and our primary outcomes. Stool toxin levels by CCA >6.4 ng/mL associated with IDSA severity (Table 1), but not the primary outcomes. Compared with the period from 2010 to 2013, the circulating ribotypes of *C. difficile* at our institution changed in 2016. Notably ribotype 106 newly emerged, accounting for 10.6% of strains, and ribotype 027 fell to 9.3% (Table 2). The incidence of ribotype 014-027 has remained stable at 18.9%, but this strain was associated with both IDSA severity and 30-day mortality (OR = 3.32; $P = 0.001$).

Conclusion. Toxin detection by EIA/CCA associated with IDSA severity, but this study was unable to confirm an association with subsequent adverse outcomes. The molecular epidemiology of *C. difficile* has shifted, and this may have implications for the optimal diagnostic strategy for CDI.

Table 1: Toxin detection vs. IDSA severity (unadjusted)

Variable	OR	95% CI	P
Toxin by EIA	1.87	1.19, 2.92	.006
Toxin >6.4 ng/mL by CCA	2.38	1.30, 4.54	.006

Table 2: Cumulative Incidence of Most Common *C. difficile* Ribotypes in the Study (2016 vs. 2010-2013)

Ribotype	2010–2013, n(%)	2016, n(%)	P
Total	1099	557	
027	181 (16.5)	52 (9.3)	<.001
014-020	178 (16.2)	105 (18.9)	.198
053-163	72 (6.6)	15 (2.7)	.001
078-126	33 (3.0)	9 (1.6)	.126
106	0 (0%)	59 (10.6)	<.001

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

2356. Increased Clinical Specificity with Ultrasensitive Detection of *Clostridioides difficile* Toxins: Reduction of Overdiagnosis Compared with Nucleic Acid Amplification Tests

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Background. *Clostridioides difficile* infection (CDI) is one of the most common healthcare-associated infections, resulting in significant morbidity, mortality, and economic burden. Diagnosis of CDI relies on the assessment of clinical presentation and laboratory tests. We have evaluated the clinical performance of ultrasensitive Single Molecule Counting technology for detection of *C. difficile* toxins A and B.

Methods. Stool specimens from 298 patients with suspected CDI were tested with nucleic acid amplification test (NAAT; BD MAX™ Cdiff assay or Xpert® *C. difficile* assay) and Singulex Clarity® *C. difficile* toxins A/B assay. Specimens with discordant results were tested with cell cytotoxicity neutralization assay (CCNA), and results were correlated with disease severity and outcome.

Results. There were 64 NAAT-positive and 234 NAAT-negative samples. Of the 32 NAAT+/Clarity− and 4 NAAT-/Clarity+ samples, there were 26 CCNA− and 4 CCNA- samples, respectively. CDI relapse or overall death was more common in NAAT+/toxin+ patients than in NAAT+/toxin− and NAAT−/toxin− patients, and NAAT+/toxin+ patients were 3.7 times more likely to experience relapse or death (Figure 1). The clinical specificity of Clarity and NAAT was 97.4% and 89.0%, respectively, and overdiagnosis was over three times more common in NAAT+/toxin− than in NAAT+/toxin+ patients (Figure 2). Negative percent agreement between NAAT and Clarity was 98.3%, and positive percent agreement increased from 50.0% to effective 84.2% and 94.1% after CCNA testing and clinical assessment.

Conclusion. The Clarity assay was superior to NAATs in diagnosis of CDI, by reducing overdiagnosis and thereby increasing clinical specificity, and presence of toxins was associated with disease severity and outcome.