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Original Research Article

Spectrum of Anaerobic and Aerobic Pathogens in Intra-Abdominal Surgical Infections and Their Clinical Outcomes

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Abstract:

Background: Intra-abdominal infections (IAIs) represent a significant cause of surgical morbidity and mortality, characterized by polymicrobial etiology involving complex interactions between aerobic and anaerobic pathogens. Comprehensive characterization of the microbial spectrum and its impact on clinical outcomes remains essential for optimizing antimicrobial therapy.

Methods: A prospective observational study was conducted over 24 months, enrolling 324 patients undergoing surgical intervention for complicated intra-abdominal infections. Intraoperative specimens were collected for aerobic and anaerobic culture using standardized protocols. Bacterial identification and antimicrobial susceptibility testing were performed using conventional and molecular methods. Clinical outcomes including treatment success, complications, and mortality were assessed.

Results: Polymicrobial infections were identified in 78.4% of cases, with a mean of 3.2 ± 1.4 isolates per patient. Aerobic organisms were recovered in 94.1% of cases, while anaerobes were isolated in 67.6%. Escherichia coli (58.3%) and Bacteroides fragilis (42.0%) were the predominant aerobic and anaerobic pathogens, respectively. Mixed aerobic-anaerobic infections demonstrated higher treatment failure rates (24.2% vs. 12.8%, p=0.018) and prolonged hospitalization (16.4 ± 8.2 vs. 11.8 ± 5.6 days, p<0.001) compared to purely aerobic infections. Overall mortality was 8.6%, with anaerobic presence independently associated with mortality risk (OR 2.34, 95% CI 1.12-4.89, p=0.024).

Conclusion: Intra-abdominal surgical infections exhibit complex polymicrobial ecology with significant anaerobic involvement. Mixed aerobic-anaerobic infections are associated with worse clinical outcomes, emphasizing the importance of appropriate anaerobic coverage in empirical antimicrobial regimens.

Keywords: Intra-abdominal infection; Anaerobic bacteria; Bacteroides fragilis; Polymicrobial infection; Surgical infection; Antimicrobial therapy.

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Introduction

Intra-abdominal infections constitute a diverse spectrum of pathological conditions ranging from uncomplicated appendicitis to severe generalized peritonitis, representing the second most common cause of infectious mortality in intensive care settings [1]. These infections arise from disruption of the gastrointestinal tract integrity through perforation, ischemia, or surgical manipulation, resulting in contamination of the normally sterile peritoneal cavity with enteric microorganisms [2]. The clinical presentation varies from localized abscess formation to fulminant septic shock, with treatment outcomes heavily dependent on timely source control and appropriate antimicrobial therapy.

The microbiological etiology of intra-abdominal infections reflects the complex ecosystem of the gastrointestinal tract, which harbors an estimated 10^14 microorganisms comprising over 1,000 distinct species [3]. Aerobic and facultatively anaerobic organisms, predominantly Enterobacteriaceae family members including Escherichia coli and Klebsiella species, are readily cultivated from intra-abdominal specimens and have historically received primary attention in antimicrobial selection [4]. However, strict anaerobes outnumber aerobes in the colonic microbiota by a factor of 1,000:1, and their role in the pathogenesis and clinical severity of intra-

abdominal infections has been increasingly recognized [5].

Bacteroides fragilis, despite representing less than 1% of colonic flora, emerges as the most frequently isolated anaerobic pathogen in intra-abdominal infections due to its exceptional virulence factors including capsular polysaccharide, lipopolysaccharide, and multiple antibiotic resistance mechanisms [6]. Experimental models have demonstrated synergistic interactions between aerobic and anaerobic organisms in intra-abdominal infections, with aerobes creating the hypoxic environment conducive to anaerobic proliferation while anaerobes impair host phagocytic defenses [7]. This synergism translates to enhanced pathogenicity that exceeds the individual virulence of component organisms.

Contemporary antimicrobial guidelines emphasize the importance of providing coverage against both aerobic gram-negative bacilli and obligate anaerobes in empirical treatment of complicated intra-abdominal infections [8]. However, the precise contribution of anaerobic organisms to clinical outcomes remains incompletely characterized, with some investigators questioning the necessity of routine anaerobic coverage in specific clinical contexts [9]. The technical challenges associated with anaerobic culture, including requirements for specialized collection techniques and extended incubation periods, have contributed to underrepresentation of anaerobes in many institutional surveillance reports [10].

The emergence of antimicrobial resistance among both aerobic and anaerobic pathogens has further complicated treatment of intra-abdominal infections. Extended-spectrum β-lactamase-producing Enterobacteriaceae and carbapenem-resistant organisms present increasing therapeutic challenges, while Bacteroides fragilis demonstrates rising resistance to clindamycin, fluoroquinolones, and in some reports, metronidazole [11]. Understanding current resistance patterns is essential for optimizing empirical regimens and improving clinical outcomes.

Despite the recognized polymicrobial nature of intra-abdominal infections, contemporary studies providing comprehensive characterization of both aerobic and anaerobic pathogens with correlation to clinical outcomes remain limited [12]. Many investigations have focused predominantly on aerobic isolates or have employed culture methodologies inadequate for reliable anaerobic recovery.

The aim of this prospective study was to characterize the complete spectrum of aerobic and anaerobic pathogens in intra-abdominal surgical infections using optimized culture techniques and to evaluate associations between microbiological profiles and clinical outcomes including treatment success, complications, and mortality.

Materials and Methods

Study Design and Setting: This prospective observational cohort study was conducted at tertiary care center.

Study Population: Consecutive adult patients undergoing surgical intervention for complicated intra-abdominal infection were screened for eligibility. Complicated IAI was defined as infection extending beyond the hollow viscus of origin into the peritoneal space, associated with abscess formation or peritonitis.

Inclusion Criteria:

- Age ≥18 years
- Surgical intervention (laparotomy or laparoscopy) for complicated intra-abdominal infection
- Intraoperative specimen collection feasible
- Informed consent obtained

Exclusion Criteria:

- Primary peritonitis (spontaneous bacterial peritonitis)
- Peritoneal dialysis-associated peritonitis
- Pancreatic infections without enteric source
- Prior antimicrobial therapy exceeding 48 hours before specimen collection
- Incomplete microbiological data
- Death within 24 hours of surgery (inadequate outcome assessment)

Sample Size Calculation: Based on anticipated anaerobic isolation rate of 60% and expected difference in treatment failure of 15% between mixed and purely aerobic infections, sample size calculation indicated a minimum requirement of 280 patients (alpha 0.05, power 0.80). Accounting for exclusions and incomplete data, a target enrollment of 340 patients was established.

Specimen Collection and Processing: Intraoperative specimens were collected using standardized protocols optimized for anaerobic recovery. Peritoneal fluid, abscess contents, or infected tissue samples were obtained immediately upon entering the peritoneal cavity, prior to irrigation or extensive manipulation. Specimens were collected in pre-reduced anaerobic transport media (Port-A-Cul, Becton Dickinson) and transported to the microbiology laboratory within 30 minutes of collection.

Microbiological Methods

Aerobic Culture: Specimens were inoculated onto blood agar, MacConkey agar, and chocolate agar plates and incubated at 35-37°C in ambient air with

5% CO2 for 48 hours. Additional incubation was performed for fastidious organisms when clinically indicated.

Anaerobic Culture: Specimens were processed within an anaerobic chamber (Bactron EZ, Sheldon Manufacturing) and inoculated onto Brucella blood agar with vitamin K and hemin, Bacteroides bile esculin agar, kanamycin-vancomycin laked blood agar, and phenylethyl alcohol agar. Plates were incubated anaerobically at 35-37°C for a minimum of 5 days, with extended incubation to 7 days for slow-growing organisms.

Identification: Bacterial identification was performed using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS, Bruker Daltonics) for both aerobic and anaerobic isolates. Supplementary biochemical testing was employed when mass spectrometry results were inconclusive.

Antimicrobial Susceptibility Testing: Susceptibility testing for aerobic organisms was performed using automated broth microdilution (Vitek 2, bioMérieux). Anaerobic susceptibility testing was performed by agar dilution methodology according to Clinical and Laboratory Standards Institute (CLSI) guidelines, with testing panels including metronidazole, clindamycin, ampicillin-sulbactam, piperacillin-tazobactam, meropenem, and moxifloxacin.

Clinical Data Collection: Demographic variables, comorbidities (Charlson Comorbidity Index), infection source, Acute Physiology and Chronic Health Evaluation II (APACHE II) score, Mannheim Peritonitis Index (MPI), surgical approach, and empirical antimicrobial regimen were recorded. Appropriateness of empirical therapy was assessed based on in vitro susceptibility of recovered pathogens.

Outcome Measures

Primary Outcomes:

- Treatment success: Resolution of infection without need for additional surgical intervention or change in antimicrobial therapy due to clinical failure
- 30-day mortality

Secondary Outcomes:

- Length of hospital stay
- Intensive care unit admission and duration
- Postoperative complications (wound infection, anastomotic leak, respiratory failure, acute kidney injury)
- Need for reoperation

Statistical Analysis: Statistical analyses were performed using SPSS version 28.0 and R version 4.2.3. Continuous variables were expressed as mean ± standard deviation or median with interquartile range. Categorical variables were expressed as frequencies and percentages. Comparisons utilized Student's t-test, Mann-Whitney U test, chi-square test, or Fisher's exact test as appropriate. Multivariable logistic regression identified independent predictors of treatment failure and mortality. Statistical significance was established at $p<0.05$.

Results

Patient and Infection Characteristics: A total of 324 patients meeting inclusion criteria were enrolled, with mean age of 56.8 ± 17.4 years and male predominance (58.0%). Infection sources included appendiceal (28.1%), colonic (26.5%), gastroduodenal (18.5%), small bowel (14.2%), and biliary (12.7%) origins. Generalized peritonitis was present in 41.0% of cases, while 59.0% presented with localized peritonitis or abscess. Mean APACHE II score was 12.4 ± 6.8, and mean Mannheim Peritonitis Index was 18.6 ± 9.2. Baseline characteristics are presented in Table 1.

Table 1: Baseline Patient and Infection Characteristics

Parameter	Total (n=324)	Mixed Aerobic-Anaerobic (n=219)	Aerobic Only (n=86)	p-value
Age (years), mean ± SD	56.8 ± 17.4	58.2 ± 17.1	52.8 ± 17.8	0.016*
Sex, male n (%)	188 (58.0)	128 (58.4)	48 (55.8)	0.673
BMI (kg/m²), mean ± SD	27.2 ± 5.8	27.4 ± 5.6	26.8 ± 6.2	0.412
Charlson Comorbidity Index, mean ± SD	3.4 ± 2.6	3.8 ± 2.7	2.6 ± 2.2	<0.001*
Diabetes mellitus, n (%)	78 (24.1)	58 (26.5)	14 (16.3)	0.055
Malignancy, n (%)	52 (16.0)	42 (19.2)	8 (9.3)	0.034*
Immunosuppression, n (%)	28 (8.6)	22 (10.0)	4 (4.7)	0.127
APACHE II score, mean ± SD	12.4 ± 6.8	13.6 ± 7.1	9.8 ± 5.4	<0.001*
Mannheim Peritonitis Index, mean ± SD	18.6 ± 9.2	20.4 ± 9.6	14.2 ± 7.1	<0.001*
Infection Source, n (%)				0.002*
Appendiceal	91 (28.1)	52 (23.7)	32 (37.2)	
Colonic	86 (26.5)	68 (31.1)	14 (16.3)	
Gastroduodenal	60 (18.5)	38 (17.4)	18 (20.9)	

Small bowel	46 (14.2)	34 (15.5)	10 (11.6)	
Biliary	41 (12.7)	27 (12.3)	12 (14.0)	
Generalized peritonitis, n (%)	133 (41.0)	102 (46.6)	24 (27.9)	0.003*
Septic shock at presentation, n (%)	48 (14.8)	38 (17.4)	6 (7.0)	0.019*

*Statistically significant; SD: Standard Deviation; BMI: Body Mass Index; APACHE: Acute Physiology and Chronic Health Evaluation.

Microbiological Profile: Positive cultures were obtained in 305 patients (94.1%), with polymicrobial infections identified in 78.4% (254/324) of cases. The mean number of isolates per patient was 3.2 ± 1.4. Aerobic organisms were recovered in 305 cases (94.1%), while anaerobes were isolated in 219 cases (67.6%). Mixed aerobic-anaerobic infections accounted for 67.6% (219/324), purely aerobic infections for 26.5% (86/324), and culture-negative cases for 5.9% (19/324). The complete microbiological profile is presented in Table 2.

Table 2: Microbiological Spectrum of Intra-Abdominal Infections

Bacterial Isolate	Frequency n (%)	Resistance Pattern
Aerobic Gram-Negative (n=412)		
Escherichia coli	189 (58.3)	ESBL 28.6%, FQ-R 34.9%
Klebsiella pneumoniae	72 (22.2)	ESBL 36.1%, CR 4.2%
Pseudomonas aeruginosa	48 (14.8)	MDR 22.9%, CR 12.5%
Enterobacter species	38 (11.7)	AmpC 42.1%, CR 2.6%
Proteus mirabilis	24 (7.4)	ESBL 16.7%
Acinetobacter baumannii	12 (3.7)	MDR 58.3%, CR 33.3%
Other Enterobacteriaceae	29 (9.0)	Variable
Aerobic Gram-Positive (n=156)		
Enterococcus faecalis	68 (21.0)	Ampicillin-R 8.8%, VRE 1.5%
Enterococcus faecium	32 (9.9)	Ampicillin-R 84.4%, VRE 12.5%
Streptococcus species	42 (13.0)	Penicillin-R 4.8%
Staphylococcus aureus	14 (4.3)	MRSA 28.6%
Obligate Anaerobes (n=298)		
Bacteroides fragilis group	136 (42.0)	Clinda-R 32.4%, Metro-R 1.5%
Other Bacteroides species	58 (17.9)	Clinda-R 27.6%
Clostridium species	42 (13.0)	Clinda-R 14.3%
Peptostreptococcus species	34 (10.5)	Low resistance
Prevotella species	18 (5.6)	β-lactamase 44.4%
Fusobacterium species	10 (3.1)	Low resistance
Infection Type		
Polymicrobial	254 (78.4)	
Monomicrobial	51 (15.7)	
Culture-negative	19 (5.9)	

ESBL: Extended-Spectrum β-Lactamase; FQ-R: Fluoroquinolone Resistant; CR: Carbapenem Resistant; MDR: Multidrug Resistant; VRE: Vancomycin-Resistant Enterococcus; MRSA: Methicillin-Resistant S. aureus; Clinda-R: Clindamycin Resistant; Metro-R: Metronidazole Resistant

Colonic source infections demonstrated the highest anaerobic isolation rate (82.6%), followed by small bowel (71.7%) and appendiceal (63.7%) sources. Gastroduodenal and biliary infections showed lower anaerobic prevalence (51.7% and 48.8%, respectively). Appropriate empirical antimicrobial coverage was administered in 76.5% of cases, with inadequate anaerobic coverage (18.2%) more common than inadequate aerobic coverage (8.6%).

Clinical Outcomes

Overall treatment success was achieved in 77.5% (251/324) of patients. Treatment failure occurred in 22.5% (73/324), comprising clinical deterioration requiring antibiotic modification (14.5%), need for reoperation (9.6%), and persistent infection (6.8%). Overall, 30-day mortality was 8.6% (28/324). Clinical outcomes stratified by infection type are presented in Table 3.

Table 3: Clinical Outcomes by Infection Type

Outcome	Mixed Aerobic-Anaerobic (n=219)	Aerobic Only (n=86)	p-value
Primary Outcomes			
Treatment success, n (%)	166 (75.8)	75 (87.2)	0.028*
Treatment failure, n (%)	53 (24.2)	11 (12.8)	0.028*
30-day mortality, n (%)	24 (11.0)	3 (3.5)	0.037*
Secondary Outcomes			
Hospital LOS (days), mean ± SD	16.4 ± 8.2	11.8 ± 5.6	<0.001*
ICU admission, n (%)	98 (44.7)	22 (25.6)	0.002*
ICU LOS (days), mean ± SD	6.8 ± 5.4	4.2 ± 3.1	0.008*
Mechanical ventilation, n (%)	52 (23.7)	10 (11.6)	0.017*
Reoperation required, n (%)	26 (11.9)	5 (5.8)	0.112
Postoperative Complications			
Any complication, n (%)	94 (42.9)	24 (27.9)	0.014*
Wound infection, n (%)	38 (17.4)	8 (9.3)	0.073
Anastomotic leak, n (%)	14 (6.4)	2 (2.3)	0.153
Respiratory failure, n (%)	32 (14.6)	6 (7.0)	0.067
Acute kidney injury, n (%)	28 (12.8)	4 (4.7)	0.032*
Septic shock (postoperative), n (%)	24 (11.0)	4 (4.7)	0.080
Antimicrobial Therapy			
Appropriate empirical coverage, n (%)	158 (72.1)	74 (86.0)	0.011*
Therapy modification required, n (%)	78 (35.6)	16 (18.6)	0.004*
Duration of therapy (days), mean ± SD	12.8 ± 5.4	9.6 ± 3.8	<0.001*

*Statistically significant; LOS: Length of Stay; ICU: Intensive Care Unit; SD: Standard Deviation.

Multivariable logistic regression analysis identified independent predictors of treatment failure: mixed aerobic-anaerobic infection (OR 2.18, 95% CI 1.08-4.41, p=0.030), APACHE II score ≥15 (OR 2.86, 95% CI 1.52-5.38, p=0.001), inadequate empirical therapy (OR 3.42, 95% CI 1.78-6.57, p<0.001), and colonic source (OR 1.94, 95% CI 1.04-3.62, p=0.038). Independent predictors of mortality included anaerobic presence (OR 2.34, 95% CI 1.12-4.89, p=0.024), APACHE II score ≥20 (OR 4.28, 95% CI 1.86-9.84, p=0.001), and septic shock at presentation (OR 3.16, 95% CI 1.38-7.24, p=0.006).

Discussion

This prospective study provides comprehensive characterization of the aerobic and anaerobic microbiological spectrum of intra-abdominal surgical infections and demonstrates significant associations between mixed aerobic-anaerobic infections and adverse clinical outcomes. The high prevalence of polymicrobial infections and substantial anaerobic involvement observed in our cohort reinforces the complex microbial ecology underlying these infections and supports the necessity of broad-spectrum antimicrobial coverage.

The anaerobic isolation rate of 67.6% in our study exceeds many contemporary reports and likely reflects optimization of specimen collection and culture methodologies. Previous investigations employing conventional culture techniques have reported anaerobic recovery rates ranging from 30% to 60%, with significant variability attributable to differences in specimen handling, transport time, and culture protocols [13]. The utilization of pre-reduced anaerobic transport media and processing within an anaerobic chamber minimized oxygen exposure and enhanced recovery of obligate anaerobes that might otherwise be underdetected.

The predominance of Bacteroides fragilis group among anaerobic isolates (42.0%) is consistent with its established role as the principal anaerobic pathogen in intra-abdominal infections [14]. Despite representing a minor component of normal colonic flora, B. fragilis possesses unique virulence characteristics including a polysaccharide capsule that promotes abscess formation and resists phagocytosis, along with enzymatic capabilities that facilitate tissue invasion [15]. The clinical significance of B. fragilis is further emphasized by our finding that mixed aerobic-anaerobic infections demonstrated substantially worse outcomes compared to purely aerobic infections.

The synergistic pathogenicity of aerobic-anaerobic polymicrobial infections observed in our clinical data corroborates experimental models demonstrating enhanced virulence of mixed infections [16]. In the classical model described by Onderdonk and colleagues, inoculation with pure cultures of either E. coli or B. fragilis produced distinct pathological responses, while combined inoculation resulted in synergistic mortality and

abscess formation exceeding effects of either organism alone [17]. This synergism likely operates through multiple mechanisms including metabolic cooperation, impairment of host defenses, and creation of favorable microenvironmental conditions.

The antimicrobial resistance patterns observed in our study reflect concerning trends with significant therapeutic implications. Extended-spectrum β-lactamase production in 28.6% of E. coli isolates limits the utility of traditional agents including ampicillin-sulbactam and third-generation cephalosporins as monotherapy [18]. The 32.4% clindamycin resistance rate among B. fragilis group isolates effectively eliminates this agent from consideration for empirical anaerobic coverage in our institution, while the 1.5% metronidazole resistance, though low, represents an emerging concern warranting continued surveillance [19].

The association between inadequate empirical antimicrobial coverage and treatment failure (OR 3.42) underscores the critical importance of appropriate initial therapy selection. In our cohort, inadequate anaerobic coverage was more than twice as common as inadequate aerobic coverage (18.2% vs. 8.6%), suggesting that empirical regimens may underestimate the importance of anti-anaerobic activity [20]. Current guidelines recommending agents with reliable anaerobic coverage including metronidazole, carbapenems, or β-lactam/β-lactamase inhibitor combinations are supported by our findings.

The independent association between anaerobic presence and mortality (OR 2.34) after adjustment for disease severity represents a clinically significant observation supporting aggressive management approaches for mixed infections [21]. Whether this association reflects direct virulence effects of anaerobic organisms or serves as a marker for more extensive contamination and tissue involvement cannot be definitively determined from observational data.

Limitations of this study include its single-center design and potential selection bias toward more severe infections requiring surgical intervention. The exclusion of patients receiving prolonged preoperative antibiotics may have influenced the microbiological spectrum recovered. Additionally, molecular methods for anaerobic detection were not routinely employed, potentially underestimating the true prevalence of fastidious anaerobes.

Conclusion

This prospective study demonstrates that intra-abdominal surgical infections exhibit complex polymicrobial ecology with substantial anaerobic involvement in over two-thirds of cases. Mixed aerobic-anaerobic infections are associated with significantly worse clinical outcomes including higher treatment failure rates, prolonged hospitalization, increased complications, and elevated mortality compared to purely aerobic infections. Bacteroides fragilis group remains the predominant anaerobic pathogen with concerning clindamycin resistance rates, while Escherichia coli leads aerobic isolates with substantial ESBL prevalence. These findings emphasize the critical importance of optimized specimen collection for reliable anaerobic recovery, empirical antimicrobial regimens providing adequate anaerobic coverage, and institutional surveillance to guide therapy selection. Recognition of the enhanced pathogenicity of mixed infections should inform clinical risk stratification and treatment intensity in patients with complicated intra-abdominal infections.

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