

Clinical Outcomes of Takotsubo Cardiomyopathy in Patients with Inflammatory Bowel Disease

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ABSTRACT

Background:

Takotsubo cardiomyopathy (TCM) and inflammatory bowel disease (IBD) have both been linked to systemic inflammatory pathways, yet the clinical relationship between these conditions remains poorly understood. We evaluated the impact of concomitant IBD on outcomes among patients hospitalized with TCM using a nationally representative database.

Methods:

Hospitalizations for TCM were identified in the National Inpatient Sample (2016–2020) and stratified according to the presence or absence of IBD. The primary outcomes were in-hospital mortality and length of stay (LOS). Secondary outcomes were cardiogenic shock, cardiac arrest, acute kidney injury (AKI), acute pulmonary edema, left ventricular thrombus, cardiac tamponade, vasopressor utilization, central venous catheter placement, and mechanical circulatory support use, including Impella and extracorporeal membrane oxygenation (ECMO). Multivariable logistic regression was performed to determine adjusted associations.

Results:

Among 40,008 TCM hospitalizations, 504 (1.3%) had concomitant IBD. Patients with IBD were younger and had fewer traditional cardiovascular risk factors. LOS was significantly longer among patients with IBD (8.6 vs 6.9 days, $p < 0.001$). Although in-hospital mortality was numerically higher, IBD was not independently associated with mortality (aOR 1.30, 95% CI 0.92–1.82; $p = 0.134$). Concomitant IBD was independently associated with increased odds of AKI (aOR 1.37, $p = 0.003$), ECMO utilization (aOR 4.21, $p = 0.006$), central venous catheter placement (aOR 1.57, $p = 0.043$), and cardiac tamponade (aOR 3.19, $p = 0.050$). Vasopressor utilization and acute pulmonary edema demonstrated nonsignificant trends toward increased risk.

Conclusion:

Patients with concomitant IBD hospitalized with TCM represent a clinically distinct subgroup characterized by greater complications and resource utilization despite fewer traditional cardiovascular risk factors. These findings suggest that chronic inflammatory disease may influence the clinical severity and phenotype of TCM and warrants further investigation.

Keywords: Takotsubo cardiomyopathy; stress cardiomyopathy; inflammatory bowel disease; acute kidney injury; systemic inflammation.

INTRODUCTION

Takotsubo cardiomyopathy (TCM), also known as stress-induced cardiomyopathy, is characterized by transient left ventricular systolic dysfunction in the absence of obstructive coronary artery disease [1]. Although previously considered benign, TCM is increasingly associated with substantial morbidity and mortality, including heart failure, arrhythmias, thromboembolic

complications, cardiogenic shock, and recurrent hospitalizations [1–3].

Growing evidence suggests that systemic inflammation plays an important role in the pathophysiology and severity of TCM. Scally et al. demonstrated persistent myocardial inflammation and elevated interleukin-6 levels in patients with TCM, suggesting inflammation as a potential contributor rather than merely a secondary response [4]. Inflammatory burden has also been

associated with adverse outcomes, including delayed ventricular recovery, increased in-hospital complications, and left ventricular thrombus formation in TCM [5,6].

Inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, is a chronic immune-mediated disorder associated with increased cardiovascular risk independent of traditional risk factors [7,8]. Proposed mechanisms include chronic systemic inflammation, endothelial dysfunction, autonomic dysregulation, and a prothrombotic state, several of which overlap with pathways implicated in TCM [9]. Emerging data regarding the "gut-heart axis" further suggest that intestinal inflammation and disruption of gut barrier integrity may contribute to myocardial inflammation and cardiovascular dysfunction [9].

Despite these shared mechanisms, the relationship between IBD and TCM remains poorly characterized. Existing evidence is limited primarily to case reports and small observational studies [10-12]. We therefore sought to evaluate clinical outcomes among patients with concomitant IBD hospitalized with TCM using a nationally representative database, with particular focus on cardiovascular complications and resource utilization

METHODS

The Nationwide Inpatient Sample (NIS), developed as part of the Healthcare Cost and Utilization Project (HCUP) and maintained by the Agency for Healthcare Research and Quality (AHRQ), is the largest publicly available all-payer inpatient database in the United States. The NIS contains hospitalization-level data, including patient demographics, hospital characteristics, diagnoses, procedures, and inpatient outcomes. As each hospitalization is recorded as a separate entry, repeat admissions for the same patient may be included. Study outcomes were identified using the International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM) coding system (Supplementary Materials, Table 1). As NIS is a publicly available deidentified database, the study was exempt from Institutional Review Board review.

We performed a retrospective cohort study using NIS data from 2016 to 2020. Adult hospitalizations (≥ 18 years) with a diagnosis of TCM were identified using ICD-10-CM diagnosis codes. Hospitalizations were stratified according to the presence or absence of concomitant IBD, including Crohn's disease and ulcerative colitis. Baseline characteristics included: age, sex, race, primary payer, median household income quartile, and hospital characteristics. Comorbidities identified included: diabetes mellitus, hypertension, dyslipidemia, overweight status, obesity, smoking history, chronic kidney disease (CKD stages 3–5), end-stage renal disease (ESRD), and coronary artery disease (CAD).

Primary outcomes were in-hospital mortality and length of stay. Secondary outcomes were cardiogenic shock,

cardiac arrest, acute kidney injury (AKI), acute pulmonary edema, mechanical ventilation, left ventricular thrombus formation, cardiac tamponade, vasopressor utilization, arterial line placement, central venous catheter placement, and use of mechanical circulatory support, including Impella and extracorporeal membrane oxygenation (ECMO).

Statistical analyses were performed using IBM Statistical Package for Social Sciences (SPSS) Statistics Software (IBM Corp., Armonk, NY, USA). Analyses were performed without application of National Inpatient Sample discharge weights. Continuous variables were expressed as means with standard deviations and compared using the independent-samples *t* test. Categorical variables were expressed as frequencies and percentages and compared using the χ^2 test. Multivariable logistic regression analyses were performed to evaluate the independent association between concomitant inflammatory bowel disease and study outcomes. Regression models were adjusted for age, sex, race, diabetes mellitus, hypertension, dyslipidemia, overweight status, obesity, smoking history, chronic kidney disease stages 3–5, end-stage renal disease, and coronary artery disease. Adjusted odds ratios (aORs) with 95% confidence intervals (CIs) were reported. A two-sided *p* value < 0.05 was considered statistically significant

RESULTS

A total of 40,008 hospitalizations with TCM were identified during the study period, of which 504 (1.3%) had a concomitant diagnosis of IBD. Patients with IBD were younger than those without IBD (64.8 ± 13.7 vs. 67.1 ± 14.2 years, $p < 0.001$), while the proportion of female patients was similar between groups (84.5% vs. 82.6%, $p = 0.249$). Racial distribution differed significantly ($p < 0.001$), with a greater proportion of White patients in the IBD cohort (88.0% vs. 80.3%) and lower proportions of Black, Hispanic, and Asian patients (Table 1).

Patients with concomitant IBD demonstrated a distinct cardiovascular risk profile. Diabetes mellitus (17.3% vs. 24.2%, $p < 0.001$), hypertension (58.9% vs. 67.0%, $p < 0.001$), and dyslipidemia (27.8% vs. 34.0%, $p = 0.003$) were less prevalent among patients with IBD. Smoking was more common in the IBD cohort (31.2% vs. 26.2%, $p = 0.012$), while obesity, chronic kidney disease, end-stage renal disease, and coronary artery disease did not differ significantly between groups. Unadjusted in-hospital mortality was numerically higher among patients with IBD but did not reach statistical significance (7.7% vs. 6.6%, $p = 0.292$). Patients with concomitant IBD had significantly longer lengths of stay than those without IBD (8.57 vs. 6.88 days, $p < 0.001$) (Tables 1 and 2).

On multivariable logistic regression analysis (Tables 3–15), concomitant IBD was not independently associated with in-hospital mortality (aOR 1.30, 95% CI 0.92–1.82, $p = 0.134$), cardiac arrest (aOR 1.05, 95% CI 0.64–1.71, $p = 0.852$), intubation (aOR 1.10, 95% CI 0.87–1.40, $p = 0.408$), or Impella utilization (aOR 0.98, 95% CI 0.24–3.98, $p = 0.980$).

IBD was not significantly associated with cardiogenic shock, although a borderline association was observed (aOR 1.36, 95% CI 0.98–1.88, $p=0.060$). No significant association was observed between IBD and left ventricular thrombus formation (aOR 1.31, 95% CI 0.53–3.19, $p=0.550$).

Patients with concomitant IBD demonstrated significantly greater odds of AKI (aOR 1.37, 95% CI 1.11–1.70, $p=0.003$) and ECMO utilization (aOR 4.21, 95% CI 1.51–11.71, $p=0.006$). Additionally, IBD was independently associated with increased central venous catheter placement (aOR 1.57, 95% CI 1.02–2.43, $p=0.043$). Arterial line placement was not significantly associated with IBD (aOR 1.50, 95% CI 0.87–2.58, $p=0.140$), while vasopressor utilization demonstrated a borderline association (aOR 1.47, 95% CI 0.99–2.17, $p=0.055$). IBD was also associated with increased odds of cardiac tamponade (aOR 3.19, 95% CI 0.998–10.19, $p=0.050$). Acute pulmonary edema was not significantly associated with IBD (aOR 1.71, 95% CI 0.91–3.23, $p=0.096$).

In multivariable analyses, increasing age was independently associated with higher in-hospital mortality (aOR 1.018 per year, 95% CI 1.015–1.021, $p<0.001$), whereas female sex was associated with lower mortality (aOR 0.45, 95% CI 0.41–0.50, $p<0.001$). Black race was associated with increased odds of mortality, cardiac arrest, intubation, and cardiac tamponade, while Hispanic and Asian race were also associated with increased mortality. Chronic kidney disease and diabetes mellitus were independently associated with multiple adverse outcomes, including mortality and AKI.

DISCUSSION

In this large nationally representative analysis, patients with concomitant IBD hospitalized with TCM represented a distinct subgroup characterized by younger age, fewer traditional cardiovascular risk factors, and increased utilization of advanced therapies. Although IBD was not independently associated with increased in-hospital mortality, it was associated with higher odds of AKI, ECMO utilization, central venous catheter placement, and cardiac tamponade, with a trend toward greater vasopressor use. Collectively, these findings support a potential association between chronic inflammatory disease and a more severe clinical presentation of TCM. To our knowledge, no prior nationwide analysis has specifically evaluated inpatient outcomes among patients with concomitant IBD and TCM. Shared mechanisms proposed between the two conditions include chronic inflammation, endothelial dysfunction, autonomic dysregulation, and psychological stress [9,10]. Our study expands upon this literature by providing large-scale epidemiologic data suggesting that patients with concomitant IBD may experience a more complicated hospital course when hospitalized with TCM.

One of the most notable findings was the significantly increased odds of ECMO utilization among patients with IBD. Although greater ECMO utilization was observed among patients with IBD, the absolute number of ECMO events was small, and this finding should therefore be interpreted cautiously. Nevertheless, it may reflect greater illness severity and the need for advanced supportive therapies in this population. The precise mechanisms underlying this association cannot be determined from administrative data; however, systemic inflammation, cytokine activation, endothelial dysfunction, and autonomic imbalance associated with IBD may amplify catecholamine-mediated myocardial dysfunction and impair compensatory cardiovascular responses during acute stress states [9]. Additionally, factors frequently encountered in IBD, including anemia, electrolyte abnormalities, and volume depletion, may further increase hemodynamic vulnerability during acute TCM presentations.

Another important finding was the increased risk of AKI among patients with IBD. This observation is clinically relevant given the multifactorial susceptibility to renal injury in IBD. Chronic inflammation, endothelial dysfunction, impaired microvascular perfusion, and recurrent episodes of volume depletion may predispose these patients to renal injury during periods of hemodynamic stress. Furthermore, AKI may serve as a marker of more severe systemic illness in TCM and could partially explain the increased resource utilization observed in the IBD cohort.

The relationship between chronic inflammatory disorders and TCM has increasingly gained attention in recent years. Several biologically plausible mechanisms have been proposed to link IBD and TCM, including chronic immune activation, endothelial dysfunction, autonomic dysregulation, and microvascular impairment [9,10]. Emerging evidence regarding the gut-heart axis further suggests that intestinal inflammation and disruption of gut barrier integrity may contribute to myocardial inflammation and cardiovascular dysfunction [9]. The present findings extend this growing body of literature by suggesting that patients with concomitant IBD may experience a more severe clinical course when hospitalized with TCM, supporting the need for further investigation into the interaction between chronic inflammatory disease and stress cardiomyopathy [10].

The increased requirement for central venous catheterization and the trend toward greater vasopressor use similarly suggest greater hemodynamic instability and resource utilization among patients with concomitant IBD and TCM. These findings are consistent with prior studies demonstrating associations between inflammatory burden and adverse outcomes in TCM. Scally et al. identified persistent myocardial inflammation in patients with TCM [4], while subsequent investigations demonstrated associations between inflammatory biomarkers and delayed ventricular recovery and other adverse clinical

outcomes [5]. Although inflammatory markers were unavailable in the present study, our findings are consistent with the hypothesis that chronic inflammatory disease may influence the severity and clinical manifestations of TCM.

Another noteworthy finding was the increased odds of cardiac tamponade among patients with IBD. Although tamponade remains a rare complication and event numbers were small, inflammatory pericardial complications have been described individually in both TCM and IBD [11,12]. These observations raise the possibility that patients with concomitant inflammatory disease may be more susceptible to pericardial involvement during TCM episodes. Nonetheless, since cardiac tamponade events were infrequent and confidence intervals were wide, this association should be interpreted cautiously and requires confirmation in future studies.

Despite increased markers of illness severity, IBD was not independently associated with increased in-hospital mortality. This may reflect the reversible nature of TCM when recognized and managed early. Similarly, there was no significant increase in LV thrombus formation despite the prothrombotic milieu associated with IBD [7,8]. The absence of association may relate to the low overall event rate and limitations inherent to administrative coding datasets.

Interestingly, patients with IBD demonstrated lower rates of hypertension, diabetes mellitus, and dyslipidemia despite comparable or greater illness severity. This observation parallels prior studies demonstrating that patients with IBD often possess fewer traditional cardiovascular risk factors while remaining at increased risk for adverse cardiovascular outcomes [13]. In our cohort, the presence of fewer conventional risk factors alongside increased complication rates and resource utilization suggests that inflammation-related mechanisms may contribute to adverse outcomes beyond traditional cardiovascular risk profiles.

This study has several limitations. As a retrospective administrative database analysis, it is subject to coding inaccuracies, residual confounding, and selection bias. The NIS lacks detailed clinical information including inflammatory biomarkers, echocardiographic findings, coronary angiography results, medication exposure, and measures of IBD disease activity. Also, as sampling weights were not applied, the findings should be interpreted within the context of the study cohort. Although application of NIS sampling weights may improve national representativeness, the observed associations remain internally valid within this large administrative database. Additionally, longitudinal outcomes, recurrent hospitalizations, and long-term ventricular recovery could not be assessed. Several complications, including cardiac tamponade and LV thrombus, were infrequent and should therefore be interpreted cautiously.

Despite these limitations, this study represents one of the largest analyses evaluating outcomes among patients with concomitant IBD and TCM. Our findings suggest that chronic inflammatory disease may influence the severity and clinical presentation of TCM and are consistent with a potential contribution of inflammatory pathways to stress cardiomyopathy. Prospective studies incorporating inflammatory biomarkers and detailed clinical data are needed to better define the relationship between IBD and TCM and to determine whether inflammatory risk stratification may improve management of this population.

CONCLUSION

In this large nationally representative analysis of patients hospitalized with Takotsubo cardiomyopathy, concomitant inflammatory bowel disease identified a clinically distinct subgroup characterized by younger age, fewer traditional cardiovascular risk factors, and increased complication burden and resource utilization. Although IBD was not independently associated with increased in-hospital mortality, patients with concomitant IBD demonstrated significantly greater odds of acute kidney injury, ECMO utilization, central venous catheter placement, and cardiac tamponade, with a trend toward increased vasopressor requirement, suggesting greater physiologic severity and higher-intensity critical care needs. These findings are consistent with the growing body of literature linking chronic inflammatory disease to adverse cardiovascular outcomes and highlight the potential relevance of the gut-heart axis in stress cardiomyopathy. To our knowledge, this study represents one of the largest investigations to date evaluating outcomes among patients with concomitant IBD and Takotsubo cardiomyopathy and provides novel evidence that chronic inflammatory disease may influence the clinical phenotype and severity of TCM. Future prospective studies incorporating inflammatory biomarkers and detailed clinical characterization are warranted to better define the mechanistic relationship between IBD and Takotsubo syndrome and to determine whether inflammation-based risk stratification may improve outcomes in this unique patient population.

CONFLICT OF INTEREST

The author declares no conflicts of interest.

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Author Note

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Table 1: Patient Demographics, Comorbidities and Unadjusted Morality

All patients Takotsubo or SCM (total n=40,008)			
Demographics	IBD	No IBD	p-value
Total n	504	39,504	
Age mean, SD	64.79, 13.69	67.09, 14.22	<.001
Sex female (%)	426 (84.5)	32,612 (82.6)	0.249
Race			<.001
White	426 (88)	30,680 (80.3)	
Black	31 (6.4)	3,142 (8.2)	
Hispanic	17 (3.5)	2,457 (6.4)	
Asian	1 (0.2)	820 (2.1)	
Native American	4 (0.8)	243 (0.6)	
Other	5 (1)	878 (0.6)	
Primary expected payer			0.044

Medicare	321 (63.7)	24,971 (63.3)	
Medicaid Private	41 (8.1) 123 (24.4)	4,276 (10.8) 8,287 (21)	
Self-pay	6 (1.2)	1,063 (2.7)	
None	2 (0.4)	78 (0.2)	
Other	11 (2.2)	784 (2)	
Population Setting			0.36
>1 million in central city	123 (24.4)	10,183 (25.9)	
>1 million fringe of city	144 (28.6)	10,075 (25.6)	
250K-1 million population	119 (23.6)	8,562 (21.8)	
50K-250K population	43 (8.5)	3,873 (9.8)	
<50K Metropolitan counties	47 (9.3)	3,893 (9.9)	
<50K Not metropolitan counties	28 (5.6)	2,766 (7)	
Median Household Income			0.537
0-25th percentile	114 (23.1)	9,888 (25.4)	
26-50th percentile	126 (25.6)	10,236 (26.3)	
51-75th percentile	137 (27.8)	9,999 (25.7)	
76-100th percentile	116 (23.5)	8,744 (22.5)	
DM	87 (17.3)	9,570 (24.2)	<.001
HTN	297 (58.9)	26,485 (67)	<.001
Dyslipidemia	140 (27.8)	13,425 (34)	0.003
Overweight BMI 25 to 29.9	10 (2)	786 (2)	0.993
Obesity BMI 30 +	56 (11.1)	4,924 (12.5)	0.36
Smoking	157 (31.2)	10,352 (26.2)	0.012
CKD 3-5	45 (8.9)	3,413 (8.6)	0.819
ESRD	8 (1.6)	805 (2)	0.476
CAD	186 (36.9)	15,715 (39.8)	0.19
Mortality	39 (7.7)	2,592 (6.6)	0.292

Table 2: Length of Stay Comparison

	IBD	No IBD	p-value
Length of Stay (mean + SD)	8.57 + 10.47	6.88+ 9.51	<.001

Table 3: Adjust Analysis of Inpatient Mortality

In-hospital mortality				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.296	0.923	1.82	0.134
Age mean (SD)	1.018	1.015	1.021	<.001
Sex female (mean)	0.452	0.412	0.496	<.001
Race				
White = REFERENCE				
Black	1.295	1.123	1.493	<.001
Hispanic	1.345	1.153	1.57	<.001

Asian	1.857	1.487	2.32	<.001
Native American	1.167	0.708	1.925	0.545
Other	1.623	1.287	2.047	<.001
DM	1.124	1.017	1.242	0.022
HTN	0.652	0.596	0.714	<.001
Dyslipidemia	0.663	0.601	0.732	<.001
Overweight BMI 25 to 29.9	0.74	0.528	1.038	0.081
Obesity BMI 30 +	0.993	0.866	1.138	0.915
CKD3-5	1.315	1.142	1.514	<.001
ESRD	1.845	1.465	2.325	<.001
Smoking	0.672	0.606	0.747	<.001
CAD	0.528	0.481	0.581	<.001

Table 4: Adjusted Analysis of Cardiac Arrest

Cardiac arrest				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.048	0.642	1.711	0.852
Age mean (SD)	0.984	0.981	0.988	<.001
Sex female (mean)	0.583	0.513	0.662	<.001
Race				
White = REFERENCE				
Black	1.587	1.34	1.88	<.001
Hispanic	1.107	0.892	1.375	0.357
Asian	1.59	1.157	2.185	0.004
Native American	1.411	0.783	2.54	0.252
Other	1.222	0.871	1.715	0.246
DM	0.983	0.854	1.131	0.81
HTN	0.845	0.746	0.957	0.008
Dyslipidemia	0.755	0.659	0.864	<.001
Overweight BMI 25 to 29.9	0.828	0.542	1.265	0.382
Obesity BMI 30 +	1.234	1.047	1.453	0.012
CKD3-5	1.126	0.906	1.401	0.284
ESRD	1.639	1.207	2.227	0.002
Smoking	0.65	0.561	0.753	<.001
CAD	0.794	0.7	0.901	<.001

Table 5: Adjusted Analysis of Cardiogenic Shock

Cardiogenic shock				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.357	0.983	1.872	0.063
Age mean (SD)	0.997	0.994	1	0.041
Sex female (mean)	0.624	0.567	0.686	<.001
Race				

White = REFERENCE				
Black	1.186	1.03	1.366	0.017
Hispanic	1.301	1.119	1.513	<.001
Asian	1.819	1.456	2.273	<.001
Native American	1.438	0.931	2.222	0.102
Other	1.119	0.865	1.447	0.394
DM	1.112	1.007	1.228	0.037
HTN	0.649	0.593	0.709	<.001
Dyslipidemia	0.811	0.738	0.892	<.001
Overweight BMI 25 to 29.9	0.943	0.702	1.268	0.7
Obesity BMI 30 +	1.133	1.001	1.284	0.048
CKD3-5	1.17	1.007	1.359	0.04
ESRD	1.514	1.187	1.932	<.001
Smoking	0.718	0.649	0.795	<.001
CAD	0.798	0.73	0.873	<.001

Table 6: Adjusted Analysis of Left Ventricular Thrombus

LV thrombus				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.308	0.537	3.185	0.554
Age mean (SD)	0.987	0.979	0.995	0.002
Sex female (mean)	0.801	0.607	1.056	0.116
Race				
White = REFERENCE				
Black	1.443	1	2.082	0.05
Hispanic	1.243	0.803	1.924	0.329
Asian	1.709	0.9	3.246	0.102
Native American	0.533	0.074	3.818	0.531
Other	1.328	0.678	2.601	0.409
DM	1	0.752	1.33	0.998
HTN	0.874	0.678	1.127	0.3
Dyslipidemia	0.771	0.59	1.009	0.058
Overweight BMI 25 to 29.9	1.614	0.822	3.166	0.164
Obesity BMI 30 +	0.779	0.53	1.144	0.203
CKD3-5	1.13	0.739	1.728	0.572
ESRD	0.601	0.222	1.631	0.318
Smoking	1.109	0.857	1.435	0.431
CAD	1.161	0.911	1.479	0.227

Table 7: Adjusted Analysis of ECMO Utilization

ECMO				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	4.209	1.513	11.71	0.006

Age mean (SD)	0.957	0.943	0.97	<.001
Sex female (mean)	0.442	0.283	0.692	<.001
Race				
White = REFERENCE				
Black	1.989	1.121	3.531	0.019
Hispanic	2.195	1.188	4.054	0.012
Asian	2.323	0.825	6.539	0.11
Native American	0	0	.	0.995
Other	1.425	0.438	4.641	0.557
DM	0.944	0.53	1.682	0.844
HTN	0.977	0.606	1.575	0.924
Dyslipidemia	0.715	0.389	1.314	0.28
Overweight BMI 25 to 29.9	2.137	0.766	5.966	0.147
Obesity BMI 30 +	1.405	0.785	2.514	0.252
CKD3-5	0.527	0.127	2.19	0.378
ESRD	1.194	0.365	3.904	0.769
Smoking	0.461	0.23	0.927	0.03
CAD	0.402	0.208	0.775	0.007

Table 8: Adjusted Analysis of AKI

AKI				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.373	1.111	1.697	0.003
Age mean (SD)	1.007	1.005	1.008	<.001
Sex female (mean)	0.434	0.408	0.461	<.001
Race				
White = REFERENCE				
Black	1.524	1.4	1.66	<.001
Hispanic	1.075	0.973	1.189	0.155
Asian	1.33	1.132	1.562	<.001
Native American	1.162	0.861	1.568	0.326
Other	1.242	1.058	1.457	0.008
DM	1.459	1.377	1.546	<.001
HTN	0.951	0.898	1.007	0.084
Dyslipidemia	0.837	0.792	0.885	<.001
Overweight BMI 25 to 29.9	1.083	0.911	1.287	0.365
Obesity BMI 30 +	1.144	1.061	1.234	<.001
CKD3-5	5.2	4.811	5.622	<.001
ESRD	1.082	0.915	1.28	0.356
Smoking	0.722	0.681	0.767	<.001
CAD	0.705	0.669	0.744	<.001

Table 9: Adjusted Analysis of Cardiac Tamponade

Cardiac Tamponade				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	3.19	0.998	10.191	0.05
Age mean (SD)	0.983	0.968	0.998	0.026
Sex female (mean)	0.747	0.449	1.244	0.262
Race				
White = REFERENCE				
Black	2.899	1.653	5.085	<.001
Hispanic	1.812	0.847	3.875	0.125
Asian	2.986	1.065	8.37	0.038
Native American	2.242	0.307	16.384	0.426
Other	1.309	0.316	5.412	0.71
DM	0.835	0.482	1.446	0.52
HTN	1.39	0.838	2.304	0.202
Dyslipidemia	0.66	0.387	1.127	0.128
Overweight BMI 25 to 29.9	2.352	0.846	6.538	0.101
Obesity BMI 30 +	1.074	0.568	2.03	0.827
CKD3-5	0.988	0.42	2.326	0.979
ESRD	1.234	0.38	4.006	0.726
Smoking	1.016	0.61	1.693	0.951
CAD	0.835	0.514	1.355	0.465

Table 10 : Adjusted Analysis of Intubation

Intubation				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.104	0.873	1.397	0.408
Age mean (SD)	0.983	0.981	0.985	<.001
Sex female (mean)	0.514	0.482	0.548	<.001
Race				
White = REFERENCE				
Black	1.323	1.206	1.45	<.001
Hispanic	1.133	1.018	1.26	0.022
Asian	1.743	1.48	2.053	<.001
Native American	1.205	0.881	1.649	0.243
Other	1.338	1.134	1.579	<.001
DM	1.143	1.069	1.222	<.001
HTN	0.7	0.659	0.743	<.001
Dyslipidemia	0.741	0.695	0.79	<.001
Overweight BMI 25 to 29.9	1.145	0.95	1.38	0.155
Obesity BMI 30 +	1.176	1.083	1.276	<.001
CKD3-5	1.102	0.991	1.224	0.072

ESRD	1.786	1.513	2.108	<.001
Smoking	0.689	0.644	0.737	<.001
CAD	0.591	0.556	0.628	<.001

Table 11: Adjusted Analysis of Arterial Line Use

Arterial line				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.503	0.874	2.582	0.14
Age mean (SD)	0.982	0.977	0.987	<.001
Sex female (mean)	0.53	0.451	0.622	<.001
Race				
White = REFERENCE				
Black	1.282	1.012	1.623	0.039
Hispanic	1.227	0.941	1.6	0.13
Asian	1.908	1.311	2.775	<.001
Native American	1.983	1.04	3.781	0.038
Other	1.289	0.847	1.961	0.236
DM	1.28	1.072	1.529	0.006
HTN	0.612	0.52	0.719	<.001
Dyslipidemia	0.926	0.777	1.105	0.395
Overweight BMI 25 to 29.9	1.137	0.695	1.861	0.609
Obesity BMI 30 +	1.056	0.845	1.32	0.631
CKD3-5	1.28	0.964	1.701	0.088
ESRD	1.687	1.128	2.523	0.011
Smoking	0.715	0.591	0.866	<.001
CAD	0.568	0.475	0.679	<.001

Table 12: Adjusted Analysis for Central Line Use

Central line				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.569	1.015	2.426	0.043
Age mean (SD)	0.992	0.988	0.996	<.001
Sex female (mean)	0.633	0.553	0.726	<.001
Race				
White = REFERENCE				
Black	1.251	1.031	1.519	0.024
Hispanic	1.399	1.135	1.723	0.002
Asian	1.82	1.334	2.484	<.001
Native American	1.014	0.497	2.069	0.97
Other	1.436	1.027	2.009	0.035
DM	1.077	0.933	1.244	0.312
HTN	0.739	0.648	0.843	<.001
Dyslipidemia	0.851	0.741	0.978	0.023

Overweight BMI 25 to 29.9	1.191	0.808	1.756	0.377
Obesity BMI 30 +	1.099	0.919	1.315	0.299
CKD3-5	1.442	1.168	1.78	<.001
ESRD	4.017	3.148	5.126	<.001
Smoking	0.76	0.655	0.883	<.001
CAD	0.572	0.498	0.657	<.001

Table 13: Adjusted Analysis for Vasopressor Use

Vasopressors				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.466	0.993	2.165	0.055
Age mean (SD)	0.996	0.992	0.999	0.014
Sex female (mean)	0.474	0.423	0.531	<.001
Race				
White = REFERENCE				
Black	1.253	1.056	1.487	0.01
Hispanic	1.294	1.073	1.561	0.007
Asian	1.949	1.492	2.545	<.001
Native American	0.983	0.518	1.864	0.957
Other	1.261	0.93	1.708	0.135
DM	1.113	0.98	1.263	0.098
HTN	0.597	0.533	0.668	<.001
Dyslipidemia	0.832	0.736	0.94	0.003
Overweight BMI 25 to 29.9	1.515	1.112	2.064	0.008
Obesity BMI 30 +	1.105	0.943	1.295	0.215
CKD3-5	1.241	1.025	1.503	0.027
ESRD	1.575	1.169	2.121	0.003
Smoking	0.701	0.615	0.8	<.001
CAD	0.54	0.478	0.611	<.001

Table 14: Adjusted Analysis for Impella Use

Impella				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	0.982	0.242	3.981	0.98
Age mean (SD)	0.995	0.984	1.006	0.394
Sex female (mean)	0.738	0.505	1.079	0.117
Race				
White = REFERENCE				
Black	0.741	0.396	1.385	0.348
Hispanic	0.919	0.493	1.713	0.789
Asian	0.812	0.257	2.565	0.722
Native American	1.702	0.417	6.949	0.459
Other	0.491	0.121	1.993	0.32

DM	1.265	0.871	1.836	0.217
HTN	0.624	0.442	0.879	0.007
Dyslipidemia	0.719	0.494	1.045	0.084
Overweight BMI 25 to 29.9	0.273	0.038	1.964	0.197
Obesity BMI 30 +	1.759	1.161	2.664	0.008
CKD3-5	1.183	0.669	2.094	0.563
ESRD	0.958	0.301	3.052	0.942
Smoking	0.489	0.313	0.763	0.002
CAD	0.967	0.689	1.356	0.845

Table 15: Adjusted Analysis for Acute Pulmonary Edema

Acute pulmonary edema				
	OR	Lower CI 95%	Higher CI 95%	p-value
IBD	1.714	0.909	3.233	0.096
Age mean (SD)	0.993	0.986	0.999	0.03
Sex female (mean)	0.929	0.734	1.175	0.539
Race				
White = REFERENCE				
Black	1.024	0.734	1.428	0.891
Hispanic	1.298	0.925	1.821	0.131
Asian	1.232	0.687	2.207	0.484
Native American	1.286	0.475	3.484	0.62
Other	1.328	0.773	2.279	0.304
DM	0.986	0.785	1.238	0.901
HTN	0.881	0.717	1.083	0.228
Dyslipidemia	0.905	0.734	1.116	0.352
Overweight BMI 25 to 29.9	1.092	0.578	2.061	0.787
Obesity BMI 30 +	0.996	0.748	1.326	0.98
CKD3-5	1.321	0.955	1.827	0.093
ESRD	2.148	1.334	3.46	0.002
Smoking	1.084	0.879	1.337	0.45
CAD	0.725	0.591	0.89	0.002

Supplementary Material:**Table 1:** ICD-10 Codes for all study outcomes

Variable	ICD-10 Codes
Takotsubo and Stress Cardiomyopathy	I5181, I4283
Inflammatory Bowel Disease (IBD)	K500, K501, K508, K509, K51
Diabetes Mellitus	E08, E09, E10, E11, E13
Hypertension (HTN)	I10
Dyslipidemia (DLD)	E780, E781, E782, E783, E784, E785

Overweight BMI 25 to 29.9 kg/m²	Z6825, Z6826, Z6827, Z6828, Z6829, E663
Obesity BMI 30 + kg/m²	Z6830, Z6831, Z6832, Z6833, Z6834, E66811, Z6835, Z6836, Z6837, Z6838, Z6839, E66812, Z684, E66813
Smoking	F17200, Z87891
Chronic Kidney Disease (CKD) Stage 3-5	N183, N184, N185
End-Stage Renal Disease (ESRD)	N186
Coronary Artery Disease (CAD)	I2510, I252, I258, I259
Intubation	0BH17EZ, 0BH18EZ, 5A1935Z, 5A1945Z, 5A1955Z
Cardiac arrest	I462, I468, I469
Arterial line	4A133B1
Cardiogenic Shock	R570
Central line	06HN33Z, 06HM33Z, 05H633Z, 05H533Z, 05HN33Z, 05HM33Z, 05HM3DZ
Left Ventricle thrombus	I513
Vasopressors	3E033XZ, 3E043XZ
Extracorporeal membrane oxygenation (ECMO)	5A1522, 5A15A2
Impella	5A0211D, 5A0221D
Acute Kidney Injury (AKI)	N17
Acute pulmonary edema	J810
Cardiac Tamponade	I314