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GENERAL SURGERY

Predictors of poor outcomes in intensive care unit patients with intra-abdominal infection

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Background: Intra-abdominal infections (IAIs) are second to respiratory-related infections as the leading cause of sepsis-related admissions to the intensive care unit (ICU). Multiple abdominal non-traumatic sources requiring intervention are responsible for these admissions. This study aims to describe the aetiology and ICU course of patients with non-traumatic intra-abdominal infections admitted to a multidisciplinary tertiary ICU. The secondary aim is to identify factors associated with mortality within this cohort of patients.
Methods: This was a single-centre, cross-sectional study conducted in a multidisciplinary ICU in a tertiary academic hospital in Johannesburg.. All patients admitted to the ICU with non-traumatic secondary and tertiary IAIs post-intervention from January 2016 to December 2020 were included. The outcomes were classified ICU discharge or mortality (early, ≤ 7 days or late, if ≥ 8 days). Univariate and multivariate logistic regression screened for mortality-related factors.
Results: A total of 390 patients met the inclusion criteria. There were multiple aetiologies of IAIs, with the dominating contributors being perforated into gastric ulcers (18%), followed by large (15%) and small (12%) bowel pathology. The primary mode of source control was laparotomy. The course of this cohort included early discharge (35%), late discharge (20%), and the overall mortality was 45.0%. The univariate and multivariate regression analysis variables associated with mortality were low albumin, elevated beta-D-glucan, low platelets, higher procalcitonin (PCT) on the day of discharge/mortality and vasopressor use.
Conclusion: The outcome of patients with IAIs admitted to the ICU is poor, with a 45% mortality. Multiple elevated biomarkers and vasopressor use were associated with mortality. This may suggest poor source control of IAI.
Keywords: intra-abdominal infection, risk factors, mortality, outcomes, intensive care unit, sepsis

Introduction

Intra-abdominal infection (IAI) is second to respiratory-related infections as the leading cause of sepsis-related admissions to an intensive care unit.[1] Unlike other types of ICU infections, the resistance rate in IAIs is similar in community-acquired compared with hospital-acquired infections (HAI). However, the one-year mortality is higher in patients with HAI and sepsis admitted to the ICU.[2,3] Various severity and prognostic scoring systems are used to identify factors associated with the risk of mortality in this patient group.[4-6]

The abdominal sepsis study (AbSeS) classification, physiological parameters for prognosis in abdominal sepsis (PIPAS) score, and a clinical prediction model for outcomes of complicated intra-abdominal infection by Ahmed et al.[7] are specific scoring systems developed for IAIs.[3,8,9] The physiological and operative severity score for the enumeration of mortality and morbidity (POSSUM) score is a good indicator of postoperative outcomes in perforation-related peritonitis. However, the newer disease-specific validated severity scoring systems for IAIs are complex and mainly used in research rather than daily clinical practice.[10-14]

The specific contributors to poor IAI patient outcomes are vast. They include advanced age, disease severity (high acute physiology assessment and chronic health evaluation (APACHE) II or high sequential organ failure assessment (SOFA) scores) on admission, prolonged time to first presentation in the hospital, inadequate source of sepsis control, associated bacteraemia, increased number of re-interventions required, deranged inflammatory markers (high C-reactive protein (CRP), high procalcitonin (PCT), low albumin) and inappropriate antibiotic use.[11,14,15]

Mortality in IAIs is also related to the development of sepsis or septic shock or both, which may lead to multiorgan failure.[16] The third international consensus defines sepsis as life-threatening organ dysfunction caused by a dysregulated host response to infection. The same group defines septic shock as a subset of sepsis in which particularly profound circulatory, cellular and metabolic abnormalities are associated with a greater risk of mortality than with sepsis alone. Patients with septic shock can be clinically identified by a vasopressor requirement to maintain a mean arterial pressure of ≥ 65 mmHg and serum lactate level greater than 2 mmol/L (> 18 mg/dL) in the absence of hypovolemia. This combination is associated with hospital mortality rates greater than 40%.[17] Mortality related to IAIs is still significantly high despite advances in diagnostic tools, surgical techniques and antibiotic use. Most of the literature on IAI outcomes is from high-income countries with different health care systems. This study aimed to describe the aetiology and ICU course of patients with non-traumatic IAIs admitted to a multidisciplinary tertiary ICU.

The secondary aim was to identify factors associated with mortality within this cohort of patients.

Methods

This single-centre retrospective, cross-sectional study reviewed the clinical records of patients postintervention, diagnosed with non-traumatic IAI. These were patients admitted between 01 January 2016 and 31 December 2020 to an 18-bed multidisciplinary ICU at Charlotte Maxeke Johannesburg Academic Hospital (CMJAH), a tertiary academic hospital with a total bed capacity of 1088. Permission to conduct the study was obtained from the University of the Witwatersrand (Wits) Human Research Ethics Committee (Medical) (M2102669). The study was conducted per the Declaration of Helsinki[18] and the South African Good Clinical Practice Guidelines.[19]

The minimum representative sample size was estimated at 150 patient records; however, all patients meeting the inclusion criteria were included in this study. A consecutive sampling method was employed for all patients meeting the inclusion criteria. The inclusion criteria for this study were: all patients 18 years and older post radiological or surgical intervention for secondary and tertiary peritonitis or IAIs. Patients initially admitted to the ward but later admitted to the ICU as a result of their IAI complications, such as relook laparotomy, were also included. The exclusion criteria in this study were: patients admitted to the general ward post-surgical intervention for IAIs who never required ICU admission, patients admitted to the ICU with primary peritonitis not requiring intervention, and patients with trauma-related secondary and tertiary peritonitis or IAIs. Documents with more than 50% missing data were also excluded.

Data collected included demographics, presence of comorbid disease, and haematological and biochemical blood results on the day of ICU admission, day seven in ICU, and the day of discharge or mortality. Other blood results collected included inflammatory markers such as CRP, PCT and albumin. The APACHE II score is used at our institution as a marker of disease severity, as it has shown an association with outcomes.[14,15] Other included parameters on data collection were the length of ICU stay, organ support required while in ICU, whether the patient was discharged early (≤ seven days) or late (≥ eight days), and early or late mortality.

Data collected were captured and kept secure in REDCap® version 11.4.4, Vanderbilt University,[20] using dedicated data-collection sheets with a study number allocated to each patient. On completion of data capturing, data were deidentified and exported into Microsoft Excel®, Version 16.5.4, Microsoft Corporation, Redmond, USA, and subsequently analysed using the R statistical software package, version 4.1.3.

Descriptive statistics were performed on continuous and categorical variables. Continuous variables found to be skewed are presented as medians and interquartile range (IQR). Categorical variables are presented in frequencies and percentages. Chi-square and Fisher's exact tests were used to test associations between severity and different parameters, such as reported comorbidities. An analysis of variance (ANOVA) was used to compare normally distributed continuous variables (e.g., APACHE II score), where the comparison was between more than two groups (early or late discharge, early or late mortality). Skewed continuous data were compared between groups using the Wilcoxon rank sum test or the nonparametric ANOVA, depending on the number of groups compared.

The logistic regression model was applied using univariate logistic regression to determine factors associated with ICU mortality, with calculated odds ratios and 95% CI. Identified associated factors with mortality from univariate analysis with $p < 0.05$ were included in the multivariate logistic regression analysis model. The factors related to outcomes were statistically significant if the p-value was less than 0.05.

Results

Recruited population demographics

There were 390 patients included in the study (Figure 1). The gender distribution was 51% females and 49% males, and the median age of all patients was 49 years (IQR 33–63).

A description of demographic variables and a comparison of survivors and non-survivors are shown in Table I. The overall mean APACHE II score was 17.4; a higher score was found in non-survivors. Older patients aged ≥ 50 years, patients requiring organ support, and those with more than one comorbid disease had reduced survival rates. Patients with known HIV status (negative or positive) had better survival (60.0% and 61.8% respectively) compared to those whose HIV status was unknown (53.0%), $p < 0.01$.

Sources of IAI

In terms of IAI source, the majority of patients admitted to ICU in this study resulted from perforated gastric/prepyloric ulcers (18%) followed by those with large bowel obstruction/perforation (15%). Pelvic inflammatory disease (PID), urosepsis and appendicitis-related IAIs had the highest survival rates (Figure 2). Laparotomy accounted for the majority of the mode of source control (94.4%), followed by radiologically assisted interventions (3.1%) and laparoscopic procedures (2.6%).

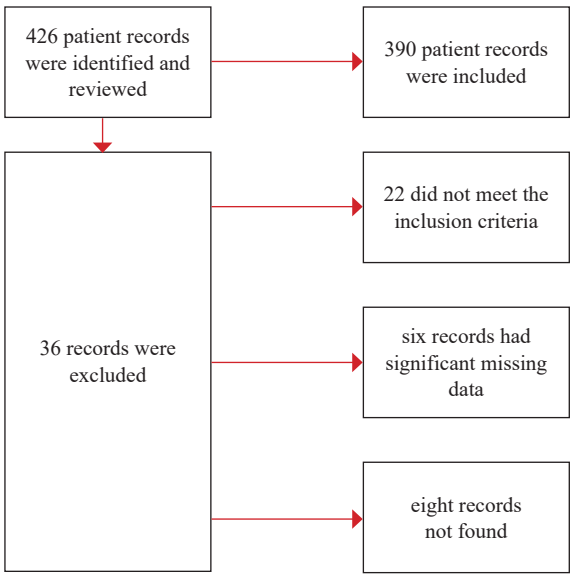

Figure 1: Study flowchart

Table I: Baseline demographic data and a comparison between survivors and non-survivors

Parameter	Total	Survivors (*n* = 213)	Non-survivors (*n* = 177)	*p*-value
	n (%) or median (IQR)			
Age (in years)	49 (33–63)	42 (30–57)	56.5 (40–67)	< 0.01
Male	191 (49.0%)	100 (47.2%)	91 (46.6%)	0.54
Female	199 (51.0%)	113 (52.8%)	86 (43.2%)	
Presence of comorbid disease:				0.02
None	169 (43.7%)	104 (48.8%)	67 (37.9%)	
One comorbidity	118 (30.5%)	66 (31.0%)	53 (29.9%)	
More than one comorbidity	100 (29.0%)	43 (20.2%)	57 (32.2%)	
HIV Status:				< 0.01
Positive	109 (27.8%)	69 (32.0%)	44 (24.9%)	
Negative	153 (39.2%)	95 (44.6%)	63 (35.6%)	
Unknown	128 (32.9%)	49 (23.0%)	70 (39.5%)	
Renal replacement during ICU admission:				< 0.01
Yes	122 (32.4%)	42 (19.8%)	80 (65.6%)	
No	255 (67.6%)	168 (79.2%)	87 (34.1%)	
Vasopressor or inotropes during ICU admission				< 0.01
Yes	248 (66.1%)	99 (46.7%)	149 (60.1%)	
No	127 (33.9%)	106 (50%)	21 (16.5%)	
Blood products transfusion:				0.19
Yes	151 (40.2%)	77 (%)	74 (49.0%)	
No	225 (59.8%)	130 (%)	95 (42.2%)	
Admission APACHE II score	17.4 (7.90–25.5)	15 (9–21)	19.5 (14.5–25.5)	< 0.01
Number of relook laparotomies	2 (1–3)	2 (1–3)	2 (1–3)	0.44
Length of ICU stay (days)	5 (3–10)	6 (3–10)	5 (2–10)	0.79

Table II: The relationship between biodata and outcomes

Parameter	Early discharge (*n* = 136)	Late discharge (*n* = 77)	P value	Early mortality (*n* = 116)	Late mortality (*n* = 61)	*p*-value
	n (%) or median (IQR)			*n* (%) or median (IQR)		
Demographics						
Age (years)	42 (30–56)	43 (32–60)	**< 0.01**	57 (42–69)	55 (40–66)	**< 0.01**
Gender: Male	63 (46.3)	37 (48.1)	0.74	56 (47.8)	35 (57.0)	0.27
Female	73 (53.7)	40 (51.9)		60 (52.2)	26 (43.0)	
Presence of comorbid disease	136 (100)	76 (100)	0.99	115 (100)	60 (100)	0.97
Admission APACHE II score	15 (6.5)	16 (9)	0.64	18.4 (7.7)	21 (7.6)	0.2
Length of ICU stay (days)	3 (2–5.5)	13 (10–21)	**<0.01**	3 (1–5)	14 (10–21)	**< 0.01**
Blood results (day 1)						
HIV positive status	38 (27.9)	30 (39.5%)	0.24	24 (20.9)	18 (30)	0.16
Hb (g/dl)	10.5 (9.2–13.1)	10.8 (9–13.7)	0.76	11.2 (9.2–13.7)	10.6 (8.7–12.2)	0.18
Platelets x10^9/L	304 (197–445)	282.5 (228.5–367.5)	0.45	270 (190–374)	274 (160–384)	0.92
Urea (mmol/L)	7.9 (4.7–13.3)	12.6 (8–17)	**< 0.01**	12.2 (8–18.9)	13 (8.2–22.8)	0.28
Creatinine (umol/L)	109.5 (76.5–188)	163.5 (96.5–282)	0.01	147 (93–230)	176 (87–290.5)	0.14
CRP (mg/L)	245 (162–350)	321 (172–350)	0.30	294 (119–350)	287 (201.5–350)	0.25
PCT (ng/mL)	14.1 (3.5–52.2)	40.4 (14.8–86)	**< 0.01**	41 (6.7–99.4)	16 (5.2–80.2)	0.17
WCC	15.2 (10.2–20.9)	14.3 (9.2–22.6)	0.76	12.5 (8.3–22.6)	13.9 (8.5–20.2)	0.93
Albumin levels day 1 (g/L)	26 (22–32.5)	22 (20–27)	**< 0.01**	25 (19–30)	23 (21–27)	0.19
Organ support						
Number of relook laparotomies	1 (1–2)	2 (1–3)	**< 0.01**	1 (1–2)	3 (1–3)	**< 0.01**
Renal replacement therapy use	17 (12.5)	25 (32.9)	**< 0.01**	38 (33.04)	42 (70.0)	**< 0.01**
Vasopressor or Inotropic support	47 (34.6)	52 (68.4)	**< 0.01**	95 (82.6)	54 (90.0)	0.38
Blood transfusions	40 (29.4)	37 (48.7)	**0.01**	38 (33.0)	36 (60.0)	**< 0.01**

ICU – Intensive care unit, PCT – Procalcitonin, Hb – Haemoglobin, CRP – C-reactive protein, WCC – White cell count

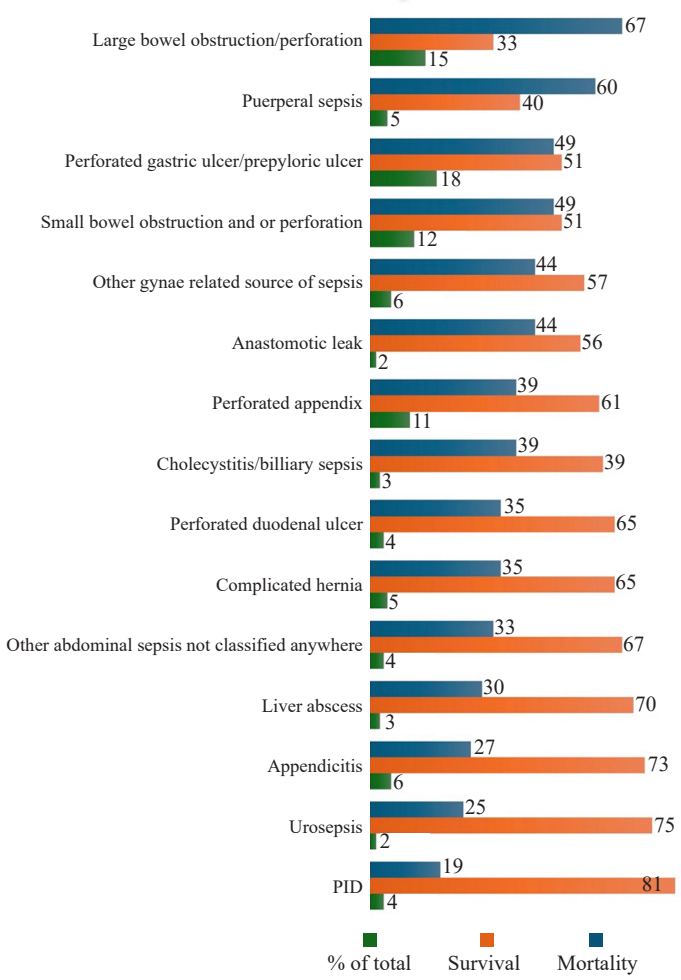

Figure 2: Distribution of sources of IAIs and frequency of survival and mortality.

Patient outcomes

The overall ICU mortality was 45.0% (n = 177). Of the patients included in this study, 35% were discharged early and 20.0% were discharged late from the ICU.

There was a significantly higher mortality in patients with IAIs resulting from large bowel obstruction/perforation (38, 66.7%) vs survivors from the same cause (19, 33.3%) ($p < 0.01$). There was no significant difference between survivors and non-survivors in patients with small bowel obstruction or perforation, or both ($p = 0.067$).-

Patients who died early (within the first week of admission) were older, mostly in their 5th decade and were largely on inotropic support (Table II). Interventions such as renal replacement therapy, and blood transfusion, were predictive of delayed mortality.

The univariate and multivariate regression models (Table III) highlight the independent effects of low albumin, low platelet count, higher PCT and beta-D-glucan, and use of vasopressors/inotropes with significantly higher odds of mortality.

Discussion

This study describes the aetiology and ICU course of patients with non-traumatic IAI admitted to a multidisciplinary tertiary ICU and identifies factors associated with mortality within this cohort of patients. Our patient ICU length of stay (LOS), which was the longest in the late mortality group with a median of 14 days and IQR (10–21), was longer compared to other studies.[21,22] In a similar study conducted in Tanzania, the median LOS was seven days (IQR 5–11), the shortest was one day and the longest was 60 days.[21]

The overall mortality in our cohort was 45.0%, which is above the average mortality quoted worldwide and in high-income countries. The overall quoted mortality from IAI worldwide is between 6.0 and 29.0%.[3,9,23] Mortality in patients with sepsis or septic shock at admission to hospital as a result of IAIs in high-income countries ranges between 7.6% and 32.4%.[24] In low- and middle-income countries (LMICs), including South Africa, the quoted mortality is between 10.0 and 74%.[11,21,25,26] Weledji et al.[27] reported disease-specific related mortality in their study; appendicitis-related mortality was around 5.0%, perforated peptic ulcer-related mortality was 25.0%, post-surgical anastomotic leaks mortality ranged between 3.0 and 22.0%, whereas colorectal perforations with soiling had a mortality of 60.0%.[27] Our study showed that patients diagnosed with IAIs secondary to large bowel obstruction/perforation had a higher likelihood of mortality. The in-between group comparison of other causes of IAIs showed no difference in outcomes, i.e., mortality and survival. However, in this study, patients with pelvic inflammatory disease as a cause of IAIs had favourable outcomes.

Factors associated with mortality in our study included advanced age, with all mortalities in patients in their 5th decade of life. Development of complications such as acute kidney injury (AKI) requiring renal replacement therapy, ongoing sepsis and septic shock, evidenced by high inflammatory markers, and use of inotropic support were also associated with mortality. Inadequate source control, suggested by multiple relook laparotomies, was more common in the mortality group.

Inadequate source control can be suspected from poor patient clinical progression, elevated inflammatory markers, worsening organ failure and positive findings from radiological investigations. Radiological investigation, such as computed tomography (CT) scans, may assist in diagnosing the source of IAI, identifying collections resulting from poor source control and managing IAI, such as using CT scan-directed percutaneous drainage.[28] As our institution had no full-time interventional radiologist at the time of the study, using percutaneous drainage when feasible was often delayed, necessitating relook surgery when clinical deterioration ensued. Laparotomy accounted for most of the mode of source control (94.4%), followed by radiologically assisted interventions (3.1%), and laparoscopic procedures accounted for the remaining 2.6%. In our setting, the main contributing factors for low laparoscopic procedures are late patient presentation, complicated abdominal sources of infection, such as elective patients now presenting with leaks, and patients presenting as emergencies with trainees doing the initial surgery. In a systematic review done in 2015, laparoscopy was recommended for treating many IAIs as it enables identification of the causative pathology and has good diagnostic accuracy.[28]

The poorly identified ongoing abdominal sepsis resulting from failed or poor source control in IAIs has been previously

Table III: Logistic regression analysis of factors associated with mortality

Variable	uOR[1] (95% CI[1])	*p*-value	aOR[1] (95% CI[1])	*p*-value
Demographics				
Age in years (>50)	1.03 (1.02, 1.05)	**< 0.001**	1.02 (1.00, 1.04)	0.076
Gender (male)	0.87 (0.58, 1.03)	0.5	0.89 (0.35, 2.21)	0.8
Number of relooks	1.07 (0.90, 1.27)	0.4	1.15 (0.90, 1.47)	0.3
Duration of symptoms in days	0.96 (0.92, 1.00)	0.063	0.93 (0.86, 1.00)	0.052
APACHE II score	1.17 (1.11, 1.23)	**< 0.001**	0.96 (0.87, 1.06)	0.4
One comorbidity	1.33 (0.70, 2.52)	0.4	0.58 (0.15, 2.23)	0.4
Two or more comorbidities	1.76 (1.15, 2.72)	**0.01**	1.55 (0.57, 4.21)	0.4
Blood results				
HIV status: Negative	Baseline		Baseline	
Positive	1.08 (0.65, 1.79)	0.8	0.93 (0.45, 1.95)	0.9
Unknown	2.38 (1.40, 4.08)	**0.001**	1.82 (0.80, 4.19)	0.2
WCC on admission	0.99 (0.97, 1.01)	0.2	0.96 (0.92, 1.00)	0.068
WCC on discharge or death	1.04 (1.02, 1.06)	**< 0.001**	1.03 (0.99, 1.08)	0.14
Albumin on admission	0.97 (0.94, 1.00)	0.023	0.97 (0.90, 1.03)	0.3
Albumin on discharge or death	0.83 (0.79, 0.88)	**< 0.001**	0.91 (0.85, 0.97)	**0.005**
Plt on admission	1.00 (1.00, 1.00)	0.15	1.00 (1.00, 1.00)	0.3
Plt on discharge or death	1.00 (1.00, 1.00)	**< 0.001**	1.00 (0.99, 1.00)	**0.008**
CRP on admission	1.00 (1.00, 1.00)	0.7	1.00 (0.99, 1.00)	0.5
CRP on discharge or death	1.01 (1.00, 1.01)	**< 0.001**	1.01 (1.00, 1.01)	0.062
PCT on admission	1.00 (1.00, 1.00)	0.11	1.00 (1.00, 1.00)	0.10
PCT on discharge or death	1.02 (1.01, 1.02)	**< 0.001**	1.01 (1.00, 1.01)	**0.007**
Beta-D-glucan	1 (1.00, 1.01)	**< 0.001**	1.01 (1.00, 1.01)	**0.01**
Urea: on admission	1.02 (1.00, 1.04)	**0.026**	0.97 (0.91, 1.04)	0.5
Urea: on discharge or death	1.09 (1.06, 1.12)	**< 0.001**	1.06 (1.00, 1.14)	0.07
Creatinine: on admission	1 (1.00, 1.00)	0.5	1 (1.00, 1.00)	0.7
Creatinine: on discharge or death	1.01 (1.00, 1.01)	**< 0.001**	1 (1.00, 1.00)	0.059
Organ support				
Renal replacement	3.68 (2.35, 5.83)	**< 0.001**	1.18 (0.57, 2.47)	0.7
Vasopressor/inotropes	7.6 (45.4, 13.2)	**< 0.001**	3.52 (1.69, 7.59)	**< 0.001**

uOR – Unadjusted odds ratio (from univariate regression model), aOR – Adjusted odds ratio (from multivariate regression model)

identified as a risk factor for mortality.[23, 29] De Pascale et al.[30] concluded that the only modifiable risk factor associated with improved odds of survival was urgent successful source control in under two hours of diagnosis; however, in the same study, empiric appropriate antibiotics did not affect survival.[30] In our setting, the referral systems from peripheral to central hospitals with adequate facilities to care for these patients may increase the time from diagnosis to source control intervention.

In this study, persistent IAI, as suggested by low albumin, use of vasopressors, elevated PCT and thrombocytopenia on the day of discharge/mortality, were independent predictors of mortality in this cohort. These markers could also suggest the presence of septic shock in the mortality group. The higher beta-D-glucan on the last day in the mortality group may suggest presence of fungal infections in these patients which also carries higher mortality, however false positive results cannot be excluded as there were no positive fungal culture results. The APACHE II score as a marker of disease severity was not predictive of mortality. The PIPAS study found that, among the ten variables included in their multivariate analysis, advanced age (> 80 years) and low platelet count were significant contributors to mortality.[9] Age as a non-modifiable risk factor was also shown to be associated with high mortality in this patient group by Arvaniti et al.[31] In their study, the four patient categories with IAI based on age were 40–59 years; young-old patients 60–69 years; middle-old patients 70–79 years and very old patients ≥ 80 years. Mortality increased with increasing age, reaching a maximum of 44.7% in the very old group.[31] A recent study by Luo et al.[15] performed in Nanfang Hospital, China, looked at factors associated with 28-day mortality in patients with IAI infections and found the SOFA score, haematocrit and fluid balance in 72 h were statistically significant in multivariate analysis.[15] Regional differences, healthcare systems and patient profiles in different countries may account for different findings in the aetiology, ICU course and outcomes in patients with IAI.

Limitations

This was a retrospective single-centre study, and the results cannot be generalised to other contexts. No data was collected on respiratory system support and ventilation challenges in these patients. We did not collect data on lactate levels and fluid status at admission, discharge or

death, both of which are known to correlate with mortality. This study only focused on ICU outcomes and not hospital or long-term outcomes of patients with IAI.

Conclusion

Outcomes of patients with IAIs admitted to the ICU are poor, with a mortality of 45.0%. The aetiology of non-traumatic IAI in our institution is multifactorial. The factors found to be independent predictors of mortality were low albumin, use of vasopressors/inotropes, low platelet count, higher PCT and beta-D-glucan, which may suggest poor source control of IAI. Further collaborative studies from low- and middle-income countries are needed to validate these findings.

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Conflict of interest

The authors declare no conflict of interest.

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Ethical approval

Ethical approval was obtained from the University of Witwatersrand (Wits) Human Research Ethics Committee (M2102669).

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