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UPPER GASTRO-INTESTINAL ENDOSCOPY

Predictors of intermediate and long-term survival in gastric variceal bleeding

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Background: Prognostic scores guide management of variceal haemorrhage, but comparative predictive data for intermediate and longterm survival in acute gastric variceal bleeding (GVB) are limited. This study evaluated the predictive value of Child-Pugh, MELD, MELD-Na and APACHE II scores for 90-day, 1, 3 and 5-year survival after acute GVB.
Methods: A retrospective cohort of 88 adults with endoscopically confirmed GVB treated between 1 January 1995 and 31 December 2024 at a tertiary referral centre was analysed. Scores were calculated from admission data. Primary endpoints were allcause mortality at 90 days, 1, 3 and 5 years. Cox proportional hazards regression and Kaplan-Meier analyses were performed.
Results: The 88 patients (56 men, 32 women), mean age 48.1 years (± 15.9), had an index admission mortality of 13.6%. Cumulative mortality at 90 days, 1, 3 and 5-years was 21.6%, 31.8%, 46.6% and 51.1%. Thresholds of Child-Pugh ≥ 10, MELD ≥ 20, MELD-Na ≥ 25 and APACHE II ≥ 20 identified patients at high risk of mortality. APACHE II was consistently associated with mortality across all time horizons (HR 1.59 at 90 days; HR 1.48 at 5 years), with the largest hazard ratios among the evaluated prognostic scores in this cohort. Clinical predictors of poor outcome included balloon tamponade, endotracheal intubation, and high transfusion requirements. Biochemical markers (hyponatraemia, hyperbilirubinaemia, elevated creatinine) further stratified risk. Patients with lower threshold scores (Child-Pugh A, MELD < 20, MELD-Na < 25, APACHE II < 20) had significantly better survival.
Conclusion: Prognostic thresholds of Child-Pugh ≥ 10, MELD ≥ 20, MELD-Na ≥ 25 and APACHE II ≥ 20 reliably stratify risk in acute GVB. APACHE II, by capturing systemic illness severity, demonstrated the strongest observed association with mortality across short- and long-term follow-up in this cohort. Integration of physiological and liver-specific scoring into clinical decision-making may improve triage, guide early salvage interventions, and enhance survival in this high-risk population.
Keywords: portal hypertension, gastric variceal bleeding, prognostic scores, survival, risk stratification

Introduction

Gastric varices (GV) occur in 20% of patients with portal hypertension either in isolation or in combination with oesophageal varices.[1] Although GVs account for only 10–15% of variceal bleeding cases, the clinical course is characteristically more severe with profuse bleeding, higher transfusion requirements and rebleeding rates, frequently requiring multiple endoscopic and salvage interventions with consequent long-term mortality rates as high as 30%.[2,3] The complexity of management relates to two inter-related issues: gastric varices differ from oesophageal varices in their vascular anatomy and blood flow haemodynamics,[4] and underlying spontaneous portosystemic shunts result in higher rebleeding rates and influence the type of endoscopic intervention required.[4] Standard endoscopy tends to underestimate true GV prevalence, making accurate endoscopic identification of high-risk subcategories crucial for optimal management.[5]

The optimal treatment for acute gastric variceal bleeding (GVB) as endorsed by the American Society of Gastrointestinal Endoscopy (ASGE),[5] the National Institute for Health and Clinical Excellence (NICE),[6] and the American Association for the Study of Liver Diseases (AASLD) practice guidelines[7] recommend targeted endoscopic control after initial resuscitation. The Sarin anatomical classification identifies different bleeding risks according to gastric location which has important clinical implications: gastric variceal band ligation is used in Gastro-Oesophageal Varices type 1 (GOV1) varices while cyanoacrylate injection is effective in GOV1 and Isolated Gastric Varices type 1 (IGV1) subcategories.[8-10] Endoscopic ultrasound-guided cyanoacrylate injection has recently emerged with improved success rates and lower rebleeding.[11] When conventional methods fail, emergency transjugular intrahepatic portosystemic shunt (TIPS) or balloon-occluded retrograde transvenous obliteration (BRTO) are salvage options, with surgical intervention as the final resort.[12-14]

Contemporary series report variable rates of initial haemostasis, recurrent bleeding and need for salvage interventions.[9,15-17] Limited consensus exists on risk

stratification for early aggressive therapy or prioritising candidates for definitive procedures. In both resource-constrained and tertiary settings, clinicians rely on prognostic scores to guide management.[18-19] The Child-Pugh (C-P) classification and Model for End-Stage Liver Disease (MELD) scores quantify hepatic dysfunction, MELD sodium (MELD-Na) incorporates hyponatraemia for refined mortality prediction, while Acute Physiology and Chronic Health Evaluation II (APACHE II) adds prognostic information in critically ill patients.[18,19] Despite routine use in oesophageal variceal bleeding, comparative data on the performance of these scores specifically in acute gastric variceal haemorrhage and their ability to predict intermediate and long-term survival are sparse.

In the absence of a dedicated prognostic score for acute GVB, this analysis evaluates the predictive values of C-P, MELD, MELD-Na and APACHE II scores for intermediate (90-day, 1 year) and long-term (3 and 5 year) survival after endoscopic intervention, in order to identify prognostic scoring threshold levels to aid risk stratification and therapeutic decision-making in this high-risk population.

Methods

This retrospective cohort study analysed consecutive adult patients with life-threatening GVB treated in the Surgical Gastroenterology Unit (SGU) at Groote Schuur Hospital and the University of Cape Town Academic Hospital in the 30-year period between 1 January 1995 and 31 December 2024. Patient follow-up data collection continued until 30 April 2026. The study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for reporting observational studies.[20] Inclusion criteria for the study were (1) age over 18 years; (2) endoscopically proven confirmation of variceal bleeding; (3) the index endoscopic intervention and all subsequent endoscopic procedures were undertaken in the SGU. Exclusion criteria for the study were (1) age under 18 years; (2) prior liver transplantation; (3) prior surgical shunt, splenectomy, or operative intervention for variceal bleeding; (4) prior TIPS, splenic embolisation or radiological intervention for variceal bleeding.

The anonymised and de-identified electronic medical information was retrieved from a prospectively maintained faculty ethics approved RedCap variceal registry. Data relating to baseline demographics, aetiology of portal hypertension, clinical and endoscopic information and biochemical variables and outcome were analysed (Table I). Sarin's classification was used to categorise the GVs.[8] C-P score and grade (A, B, or C), MELD, MELD-Na scores and APACHE II scores were calculated and used to grade the severity of the underlying liver disease. Patient age, sex, aetiology of cirrhosis, concomitant hepatocellular carcinoma, C-P grade, presentation of active bleeding, type of GVs, C-P score and MELD scores were considered as potential confounders for comparison.

Table I: Baseline characteristics of the cohort according to 90-day mortality status

Risk Factors	Total cohort (n = 88)[1]	Died < 90d (n = 19)[1]	Survived ≥ 90d (n = 69)[1]	p-value[2]
Age, mean (SD)	48.1 (15.9)	50.7 (16.4)	47.4 (15.9)	0.4
Male	56 (63.6%)	12 (63.2%)	44 (63.8%)	> 0.9
Female	32 (36.4%)	7 (36.8%)	25 (36.2%)	
Units blood transfused, median (range)	4.0 (4.0–6.0)	6.0 (6.0–8.0)	4.0 (4.0–5.0)	< 0.001
Balloon tube: Yes	7 (8.0%)	7 (36.8%)	0 (0.0%)	< 0.001
Balloon tube: No	81 (92.0%)	12 (63.2%)	69 (100.0%)	
Endotracheal intubation: Yes	9 (10.2%)	9 (47.4%)	0 (0.0%)	< 0.001
Endotracheal intubation: No	79 (89.8%)	10 (52.6%)	69 (100.0%)	
Na ≤ 130 mmol	23 (26.1%)	15 (78.9%)	8 (11.6%)	< 0.001
Na > 130 mmol	65 (73.9%)	4 (21.1%)	61 (88.4%)	
Creatinine ≥ 120 µmol/L	11 (12.5%)	11 (57.9%)	0 (0.0%)	< 0.001
Creatinine < 120 µmol/L	77 (87.5%)	8 (42.1%)	69 (100.0%)	
Bilirubin ≥ 51 µmol/L	22 (25.0%)	13 (68.4%)	9 (13.0%)	< 0.001
Bilirubin < 51 µmol/L	66 (75.0%)	6 (31.6%)	60 (87.0%)	
Child-Pugh A	27 (30.7%)	0 (0.0%)	27 (39.1%)	< 0.001
Child-Pugh B	34 (38.6%)	4 (21.1%)	30 (43.5%)	
Child-Pugh C	27 (30.7%)	15 (78.9%)	12 (17.4%)	
Child-Pugh score < 10	61 (69.3%)	4 (21.1%)	57 (82.6%)	< 0.001
Child-Pugh score ≥ 10	27 (30.7%)	15 (78.9%)	12 (17.4%)	
MELD < 20	75 (85.2%)	7 (36.8%)	68 (98.6%)	< 0.001
MELD ≥ 20	13 (14.8%)	12 (63.2%)	1 (1.4%)	
MELD-Na < 25	75 (85.2%)	7 (36.8%)	68 (98.6%)	< 0.001
MELD-Na ≥ 25	13 (14.8%)	12 (63.2%)	1 (1.4%)	
APACHE II < 20	78 (88.6%)	10 (52.6%)	68 (98.6%)	< 0.001
APACHE II ≥ 20	10 (11.4%)	9 (47.4%)	1 (1.4%)	

[1]Mean (±SD), n (%), Median (Q1–Q3)
[2]Wilcoxon rank sum test, Pearson's chi-square test, Fisher's exact test

Details of the acute bleeding management protocol and the endoscopic interventional techniques used in our unit have been published in detail previously.[21-23] If bleeding gastric varices were encountered, injection sclerotherapy (IST) with ethanolamine oleate or endoscopic variceal band ligation (EVL) or histoacryl/cyanoacrylate glue injection was used depending on the location and size of the varix.[23] In patients with uncontrollable bleeding a Sengstaken-Blakemore balloon tube was inserted for tamponade.[24] Endotracheal intubation was used for airway protection when necessary and further endoscopic procedures were performed within 24 hours.[23] In patients who had uncontrolled variceal bleeding or those with persistent refractory bleeding despite endoscopic intervention, radiological portosystemic shunting or salvage surgery was used as a rescue treatment. After discharge from hospital, patients underwent endoscopic follow-up after one month, three months and then every six months.

The primary end points of the study were 90-day, one-, three-, and five-year mortality. Primary sub-analysis differentiated between liver-related and non-liver-related deaths. Mortality due to haemorrhage (gastrointestinal bleeds), liver decompensation (encephalopathy, renal failure), or malignancy (HCC) were defined as liver-related deaths. Non-liver-related deaths were classified as death attributed to infection/s (sepsis, pneumonia, spontaneous bacterial peritonitis), cardiovascular disease or non-hepatic malignancy. Secondary endpoints were initial control of gastric variceal bleeding, subsequent rebleeding and the need for primary or secondary salvage procedures. All-cause mortality data was retrieved from the Faculty RedCap variceal registry and the national mortality database.

Statistical methods

The characteristics of the patients were summarised as median (interquartile range (IQR)) or mean (± standard deviation) for continuous variables and numbers (percentages) for categorical data. Continuous variables were compared with independent t-tests, analysis of variance (ANOVA), or the nonparametric Mann-Whitney rank-sum test, as appropriate. Categorical variables were analysed with Pearson's chi-square test or Fisher's exact test as appropriate. All tests were two-sided. Time-to-event analyses were performed using Cox proportional hazards regression to evaluate prognostic factors for short- and long-term outcomes following variceal bleeding. Survival time was defined as the number of days from the index bleed to death or last follow-up. Given the limited number of deaths during follow-up and the overlap among liver-specific prognostic scores, multivariable Cox regression was not performed to avoid overfitting and collinearity. Instead, separate Cox proportional hazards models were fitted for C-P grade, C-P score, MELD score, MELD-Na score, and APACHE II score at four predefined time horizons (90 days, 1 year, 3 years, and 5 years following the index bleed). Patients who remained alive beyond each time horizon were administratively censored at that time point. C-P grade was entered as a categorical variable with C-P A as the reference group. Hazard ratios (HRs) with 95% confidence intervals (CIs) and *p*-values were reported. A *p*-value of < 0.05 was considered statistically significant. All statistical analyses were performed using R statistical software (R Foundation for Statistical Computing, Vienna, Austria), version 4.6.0.

The study protocol was approved by the Human Research Ethics Committee (HREC Ref No. 710/2021) of the University of Cape Town, and the research was conducted in accordance with the Declaration of Helsinki.

Results

A total of 626 patients with variceal bleeding were admitted to the SGU during the study period. In 88 patients (14.1%) bleeding was due to gastric varices, and this cohort forms the study population. Fourteen (15.9%) of the 88 patients had previously received endoscopic treatment for oesophageal variceal bleeding. The 56 (63.6%) men and 32 women had a mean age of 48.1 years (±15.9). Cirrhosis was caused by alcohol in 44 (50%) patients, hepatitis B virus (HBV) in 9 (10.2%), both alcohol and HBV in 7 (7.9%), metabolic dysfunction-associated steatotic liver disease (MASLD) in 6 (6.8%), primary sclerosing cholangitis (PSC) in 4 (4.5%), bilharzia in 3 (3.4%), cryptogenic cirrhosis in 3 (3.4%) and haemochromatosis in 1 (1.1%). Two patients had portal hypertension and gastric varices due to congenital hepatic fibrosis (2.3%) and nine had portal or splenic vein thrombosis (10.2%). Cirrhosis in three patients was complicated by metastatic hepatocellular carcinoma (HCC) and three further patients at presentation had, in addition to cirrhosis, advanced human immunodeficiency virus (HIV) (World Health Organization stage ≥ 3, CD4 cell count < 200 cells/mm^3). The type of GV was GOV1 in 50 patients (56.8%), GOV2 in 31 patients (35.2%) and IGV1 in 7 patients (8.0%).

Endoscopic intervention in the 88 patients on 265 occasions during the index admission included glue injection in 53 patients (60.2%), endoscopic variceal ligation (EVL) in 21 patients (23.9%), injection sclerotherapy (IST) with ethanolamine oleate in 9 patients (10.2%) and EVL+IST in 5 patients (5.7%) (glue $n = 135$, EVL $n = 72$, IST $n = 46$, EVL+IST $n = 12$). Median units of blood transfused were 4.0 (IQR 1.0–6.0). Nine patients required endotracheal intubation, ventilation and inotropic support during resuscitation and endoscopic intervention. Salvage intervention for intractable or recurrent bleeding during the index admission included balloon tamponade ($n = 7$, 8.0%) and TIPS ($n = 1$, 1.1%) and surgical intervention with a gastric devascularisation ($n = 2$, 2.3%). The endoscopic failure rate of haemostasis during the index admission was 9.1% (8/88 patients). Forty-two (47.7%) patients in addition to gastric varices, also required endoscopic intervention for bleeding oesophageal varices during the index admission (EVL+IST $n = 20$; EVL $n = 18$, IST $n = 4$).

Twelve of the 88 patients (13.6%) died in hospital during the index admission (median 7.5, range 1–51 days) of uncontrolled GVB $n = 3$, adult respiratory distress syndrome $n = 3$, liver failure $n = 2$, multi-organ failure $n = 1$, metastatic hepatocellular carcinoma $n = 1$, sepsis due to candidaemia $n = 1$, and a cerebrovascular event $n = 1$. In the 12 patients who died during the index admission the mean C-P score was 11.6 (± 1.9), mean MELD score was 22.3 (± 4.2) and the mean MELD-Na score was 26.7 (± 5.7). Mortality in C-P grade A was 0%, C-P grade B 8.8% and C-P grade C 33.3%.

During the study period the 88 patients underwent a total of 780 endoscopic procedures (range 1–34, mean 5), 265 during the index admission and 515 procedures during subsequent follow-up. Gastric varices were eradicated in 47 (77%) of 61 patients alive at 1-year during follow-up

Table II: Kaplan-Meier estimated survival probabilities according to prognostic factors in 88 patients with bleeding gastric varices

Score	*n*	90-day survival	1-year survival	3-year survival	5-year survival	Log-rank *p*-value
Child-Pugh A	27	100.0%	100.0%	84.2%	79.8%	
Child-Pugh B	34	88.2%	73.5%	58.8%	55.4%	
Child-Pugh C	27	44.4%	29.6%	14.8%	4.9%	< 0.001
Child-Pugh score < 10	61	93.4%	85.2%	69.9%	66.0%	
Child-Pugh score ≥ 10	27	44.4%	29.6%	14.8%	4.9%	< 0.001
MELD < 20	75	90.7%	78.7%	62.1%	55.7%	
MELD ≥ 20	13	7.7%	7.7%	0.0%	0.0%	< 0.001
MELD-Na < 25	75	90.7%	78.7%	62.1%	55.7%	
MELD-Na ≥ 25	13	7.7%	7.7%	0.0%	0.0%	< 0.001
APACHE II < 20	78	87.2%	75.6%	59.7%	53.6%	
APACHE II ≥ 20	10	10.0%	10.0%	0.0%	0.0%	< 0.001

*Values are Kaplan-Meier estimated survival probabilities at each follow-up time point.
**Values represent estimated numbers of patients surviving at each time point based on Kaplan-Meier survival estimates

endoscopy. Varices recurred in 5 patients after eradication and rebleed in 1 patient after eradication. Seventeen patients rebled before eventual endoscopic eradication of gastric varices. Endoscopic intervention failed to control GVB in 20 patients, 4 during the initial endoscopy, and in 16 patients as a secondary event during the eradication process.

Ultimately, during the full long-term study period 17 (19.3%) patients required a salvage procedure for endoscopically uncontrolled GVB. Salvage procedures included TIPS (*n* = 5), BRTO (*n* = 4), gastric devascularisation and splenectomy (*n* = 3), oesophageal transection and gastric devascularisation (*n* = 2), gastric devascularisation alone (*n* = 1), Warren distal splenorenal shunt (*n* = 1) and an Inokuchi selective portosystemic shunt between the coronary vein and the inferior vena cava (*n* = 1). Three patients who underwent an emergency salvage procedure (devascularisation *n* = 2, TIPS *n* = 1) during the index admission died due to liver failure (*n* = 2) and candidaemia (*n* = 1). Thirteen of the 14 remaining salvage interventions are alive as of 30 April 2026: BRTO (*n* = 4, mean 2.5 years, range 1–4 years), TIPS (*n* = 4, mean 6.0 years, range 1–19 years), devascularisation (*n* = 3, mean 12 years, range 2–16 years), and shunts (*n* = 2, mean 17 years, range 6–29 years).

Long-term follow-up showed substantial morbidity which included encephalopathy (*n* = 3) after TIPS which resolved on treatment, Klebsiella bacterial peritonitis (*n* = 3) which required IV antibiotics, aspiration pneumonia (*n* = 2) and COVID pneumonia (*n* = 2). Three patients had postoperative complications, wound sepsis (*n* = 2) and one patient had a partial wound sheath dehiscence which required resuturing. Seven patients had oesophageal sequelae after interventional endoscopy which included oesophageal mucosal ulceration (*n* = 3), oesophageal stricture which resolved with dilatation (*n* = 3), and one patient developed candida oesophagitis.

Index admission hospital mortality was 13.6% (12/88), 6-week mortality 15.9% (14/88), 90-day mortality 21.6% (19/88), 1-year overall mortality 31.8% (28/88), 3-year mortality 46.6% (41/88), and 5-year mortality was 51.1% (45/88). The leading causes of overall mortality at 5 years after discharge from hospital (*n* = 31) were progressive liver failure (*n* = 13) predominantly in C-P C patients with advanced decompensated cirrhosis, metastatic hepatocellular carcinoma in 5 patients, stage ≥ 3 HIV in 3 patients, uncontrolled recurrent oesophageal (*n* = 4) and gastric (*n* = 3) variceal bleeding and acute respiratory distress syndrome (ARDS) in 3 patients.

Table III: Univariable Cox proportional hazards models for all-cause mortality

Outcome	Variable	HR	95% CI	*p*-value
90-day survival	Child-Pugh score	2.01	1.54–2.63	< 0.001
	MELD	1.28	1.18–1.38	< 0.001
	MELD-Na	1.26	1.16–1.36	< 0.001
	APACHE II	1.59	1.38–1.82	< 0.001
1-year survival	Child-Pugh score	1.76	1.47–2.12	< 0.001
	MELD	1.25	1.17–1.34	< 0.001
	MELD-Na	1.23	1.15–1.30	< 0.001
	APACHE II	1.51	1.36–1.68	< 0.001
3-year survival	Child-Pugh score	1.59	1.38–1.82	< 0.001
	MELD	1.24	1.17–1.31	< 0.001
	MELD-Na	1.20	1.14–1.26	< 0.001
	APACHE II	1.46	1.34–1.59	< 0.001
5-year survival	Child-Pugh score	1.60	1.40–1.83	< 0.001
	MELD	1.24	1.17–1.31	< 0.001
	MELD-Na	1.20	1.14–1.26	< 0.001
	APACHE II	1.48	1.36–1.61	< 0.001

CI – confidence interval, HR – hazard ratio

Overall survival rates at 90 days, 1-year, 3-years and 5-years were 78.4%, 68.2%, 53.4% and 48.9% respectively. On bivariate analysis, clinical characteristics which were significantly associated with 90-day mortality ($p < 0.001$) were ≥ 6 units of blood transfused, balloon tube placement and requirement for endotracheal intubation, while significant biochemical factors ($p < 0.001$) were Na ≤ 130 mmol/L, creatinine ≥ 120 µmol/L, bilirubin ≥ 51 µmol/L. Prognostic scoring factors significantly associated with 90-day mortality ($p < 0.001$) were C-P score ≥ 10, MELD ≥ 20, MELD-Na ≥ 25 and APACHE II score ≥ 20 (Table I). Ninety-day mortality for C-P grade C patients was 55.6%, for C-P score ≥ 10 48.4%, MELD score ≥ 20 91.7%, MELD-Na score ≥ 25 91.7%, and APACHE II score ≥ 20 90% (Table II). The MELD ≥ 20 and MELD-Na ≥ 25 groups each included only 12 patients. Consequently, the corresponding survival estimates were less precise, with

Table IV: Kaplan-Meier survival probabilities according to gastric variceal type

GV type	*n*	6-week survival	90-day survival	1-year survival	3-year survival	5-year survival	Log-rank *p*-value
GOV1	50	86.0%	78.0%	66.0%	45.6%	40.8%	
GOV2	31	80.6%	77.4%	67.7%	57.6%	49.9%	
IGV1	7	85.7%	85.7%	85.7%	85.7%	85.7%	0.10

*Values are Kaplan-Meier estimated survival probabilities at each follow-up time point

wider confidence intervals, and should be interpreted with caution.

Patients with C-P grade A, C-P score < 10, MELD score < 20, MELD-Na score < 25 and APACHE II scores < 20 had significantly better 90-day, 1, 3 and 5-year survival rates (Table II). Univariable Cox proportional hazards models demonstrated significant associations between APACHE II and mortality at 90 days, 1 year, 3 years and 5 years (HR 1.59, 1.51, 1.46 and 1.48, respectively). At 90 days, MELD and MELD-Na were also significantly associated with an increased hazard of death (HR 1.28 and 1.26, respectively) (Table III). Six-week survival of gastric variceal types GOV1, GOV2 and IGV1 were similar (86.0, 83.9, 85.7%) (Table IV). Although IGV1 appeared to have higher estimated 5-year survival rates than those with GOV1 or GOV2 (85.7% vs. 40.8 and 49.9% respectively), the overall survival curves did not differ significantly among the three gastric variceal types (log-rank test, $p = 0.1$).

Discussion

This study represents the largest single-centre series of GVB in sub-Saharan Africa and demonstrates three key findings. First, endoscopic haemostasis was achieved in most patients, yet in-hospital mortality remained high (13.6%), reflecting the severity of underlying liver disease and systemic compromise. Second, survival declined progressively after discharge, with five-year mortality approaching 50%, consistent with international data where in-hospital mortality for GVB ranges from 10–20% and five-year mortality approaches 50%.[1,3,5-7,15] The leading causes of death, progressive liver failure, hepatocellular carcinoma, HIV-related complications, and recurrent variceal bleeding reflect the multifactorial nature of mortality in cirrhotic patients.[18,19] Thirdly, this study identified clinically useful thresholds of C-P ≥ 10, MELD ≥ 20, MELD-Na ≥ 25 and APACHE II ≥ 20 that were associated with increased mortality. Mortality was especially pronounced at the index admission in C-P C patients, in whom the death rate was 33.3%, compared to 0% in C-P A patients. These results are concordant with large multicentre studies demonstrating that the C-P grade remains a consistent and powerful predictor of mortality following any variceal bleeding event.[19,25,26] These findings reinforce the prognostic utility of established scoring systems in GVB, consistent with global literature.[18,19] Although mortality rates in this cohort are comparable to those reported in Europe and Asia, the contribution of HIV and bilharzia to underlying liver disease is geographically distinctive highlighting the need for region-specific strategies that address the unique epidemiology of portal hypertension in Africa.

The study also highlights several clinical and biochemical predictors of poor outcome. Balloon tamponade, endotracheal intubation, and high transfusion requirements were strongly associated with index admission mortality. Biochemical markers including hyponatraemia, hyperbilirubinaemia, and elevated creatinine further stratified risk. These findings mirror international reports that emphasise the prognostic importance of renal dysfunction, electrolyte imbalance, and transfusion burden in variceal bleeding.[18,19,26] The MELD family of scores has gained wide acceptance in predicting mortality in variceal bleeding.[18,19,24] In the current cohort, MELD ≥ 20 and MELD-Na ≥ 25 scores demonstrated high hazard ratios for mortality, suggesting that these thresholds may be useful in identifying patients requiring more aggressive support or earlier consideration for TIPS. However, the MELD ≥ 20 and MELD-Na ≥ 25 groups each comprised only 12 patients; therefore, the corresponding survival estimates should be interpreted with caution because of their reduced precision. The strong association between hyponatraemia and mortality reflected in the MELD-Na score is particularly noteworthy. Hyponatraemia reflects severe portal hypertension, systemic vasodilation, and impaired renal perfusion and has been identified as a major determinant of post-haemorrhage outcomes in multiple studies.[18,19,26] Univariable Cox regression demonstrated APACHE II had the largest hazard ratios for mortality, particularly at 90 days, indicating the strongest association with mortality among the prognostic scores evaluated. As this study did not formally compare model discrimination or calibration, these findings should be interpreted as demonstrating a stronger association with mortality rather than definitive superiority of APACHE II as a prognostic model. This finding supports earlier evidence that variceal bleeding mortality is strongly influenced by extrahepatic organ dysfunction, particularly respiratory and renal failure, and that critical care scoring systems may complement liver disease assessment in emergency triage.[19,26]

The reported mortality associated with acute GVB varies widely and is dependent on the availability of an integrated skilled endoscopy service experienced with cyanoacrylate injection and a proficient radiological intervention team offering salvage decompression treatment.[19,20] Globally, cyanoacrylate injection remains the gold standard for control of GVB, with haemostasis rates exceeding 80%.[5,15,16] In this study the failure rate of 9.1% is within expected ranges and the acute haemostasis rate was comparable to or better than most series of patients treated with a tissue adhesive.[9,11] Differences observed between studies may reflect different injection techniques, different proportions of GV subtypes, and also inclusion of different patient populations.[19,20] Some studies had a preponderance of noncirrhotic PHT, whereas in other studies more patients had cirrhosis and associated hepatic synthetic dysfunction.[9,11,26,27] Sarin et al.[8] reported that endoscopic success was weighted heavily by high efficacy in GOV1 (95% eradication) and was less effective for GOV2 and IGV1. In that study, rebleeding after elective EVS was less than 20% for patients with GOV1 and GOV2 but higher in patients with IGV1 (53%).[8]

In the present study, patients with IGV1 demonstrated the highest estimated 5-year survival; however, this difference did not reach statistical significance. This finding should

be interpreted with caution as there were only 7 patients in the IGV1 group and 1 death. The GOV2 subgroup was also relatively small compared with the GOV1 subgroup. The limited sample size and small number of events reduced the statistical power of the log-rank test, increasing the possibility of a type II error. Consequently, the absence of a statistically significant difference does not necessarily indicate equivalent long-term survival among the gastric variceal subtypes.

The data documented must be interpreted in the light of inherent limitations which include the long duration of the study. In addition, there inevitably will be a selection bias as the unit functions as a tertiary referral centre for high-risk patients. Management practices have evolved over the three decades, especially the availability of TIPS and BRTO, although core protocols remained consistent. Multivariable Cox regression was not performed due to the limited number of deaths and overlap among the prognostic scores which would have resulted in overfitting and collinearity; consequently, the reported associations should not be interpreted as independent effects. Additionally, this is a single-centre study, and results may not be generalisable to settings with different resources or expertise. The major strengths of this study include the sample size, a prospectively maintained registry, uniform management protocols, and detailed clinical and biochemical data. Few centres worldwide have comparable long-term experience managing GVB in a single specialised unit, making this dataset particularly valuable. A further strength is the analysis of real-world data from an unselected cohort of consecutively treated patients by the same lead author using specific endpoints.

The findings of this study have several important implications for patients with major GVB that cannot be controlled endoscopically. This high-risk group is best managed in a tertiary referral unit with a skilled multidisciplinary team. Patients in whom endoscopic intervention fails to control bleeding should be identified early so that TIPS or BRTO can be performed before the deleterious consequences of deteriorating liver function and multi-organ failure occur. Prognostic scores such as CP, MELD, MELD-Na, and APACHE II should be routinely applied to guide management decisions and identify patients at high-risk of dying. Endoscopic glue injection remains the mainstay of therapy, but training and resource allocation should be prioritised to ensure its widespread availability.

Conclusion

This study provides valuable insights into the management and outcomes of GVB in South Africa. The findings emphasise the importance of early risk stratification, aggressive resuscitation within evidence-based protocols, and timely referral for salvage procedures. The data demonstrate that mortality after acute GVB is driven primarily by the severity of underlying liver disease and acute physiological instability, rather than failure of endoscopic haemostasis alone. Prognostic scores remain essential tools with the C-P, MELD, MELD-Na, and APACHE II scores all providing meaningful risk stratification. APACHE II demonstrated the strongest association with mortality in this cohort, although formal comparisons of predictive performance were not undertaken. Expanding access to advanced salvage interventions such as TIPS and BRTO, alongside integrated management of comorbidities, offers the best opportunity to improve survival in this high-risk population.

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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 Cape Town, Faculty of Health Sciences, Human Research Ethics Committee (Ref: 710/2021).

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