The Unique Impact of Blood Type O on Gastrointestinal Bleeding with Left Ventricular Assist Systems

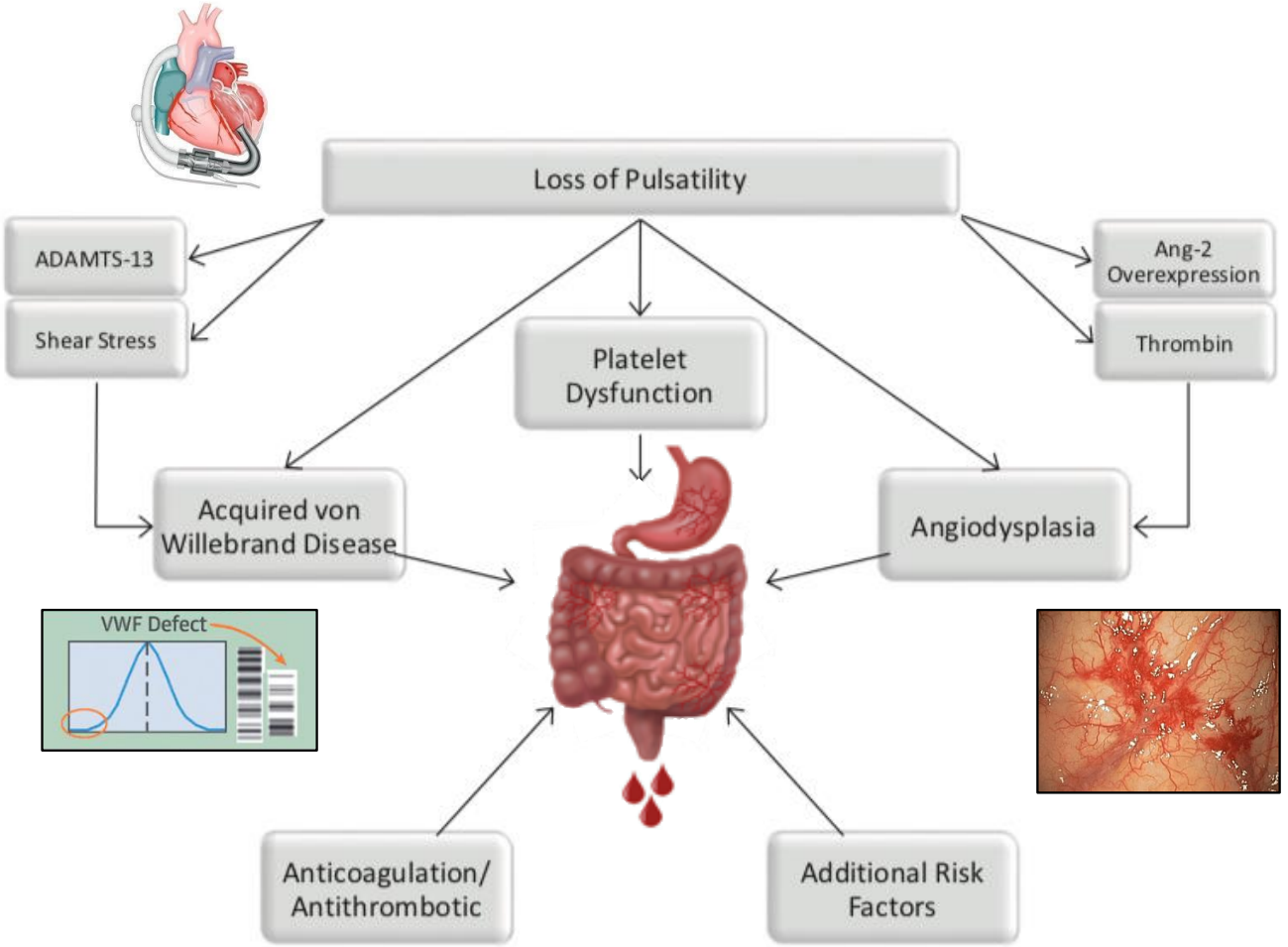
Jefferson L Vieira MD, PhD, Michael Pfeffer MD, Brian Claggett PhD, Jean M Connors MD, Garrick C Stewart MD, MPH, Michael M Givertz MD, Lara Coakley NP, Hari R Mallidi MD, and Mandeep R Mehra MD, MSc

HARVARD MEDICAL SCHOOL
TEACHING HOSPITAL

MAGNET RECOGNIZED
AMERICAN NURSES CREDENTIALING CENTER

Disclosures: None

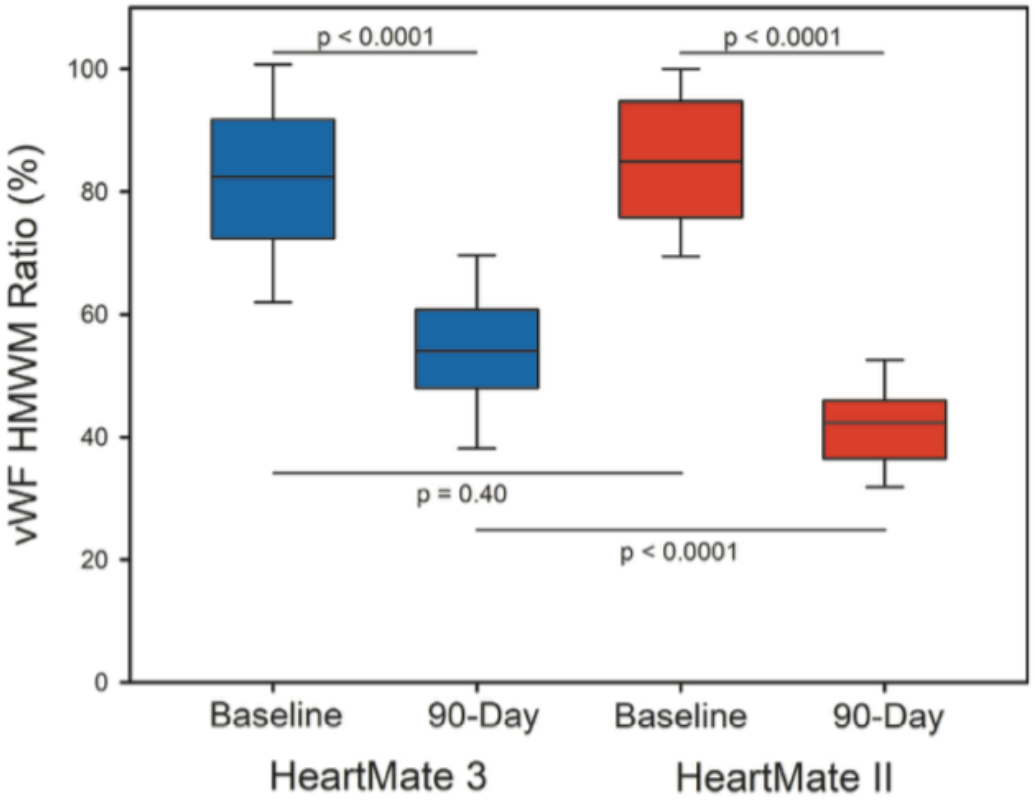
The genesis of gastrointestinal bleeding in patients with CF-LVAS s is likely multifactorial



Adapted from Eric Van Belle et al. JACC. 2019 and
Adam C. Sieg et al. Pharmacotherapy 2017

Both centrifugal and axial-flow pumps exhibit degradation of high-molecular-weight von Willebrand factor multimers

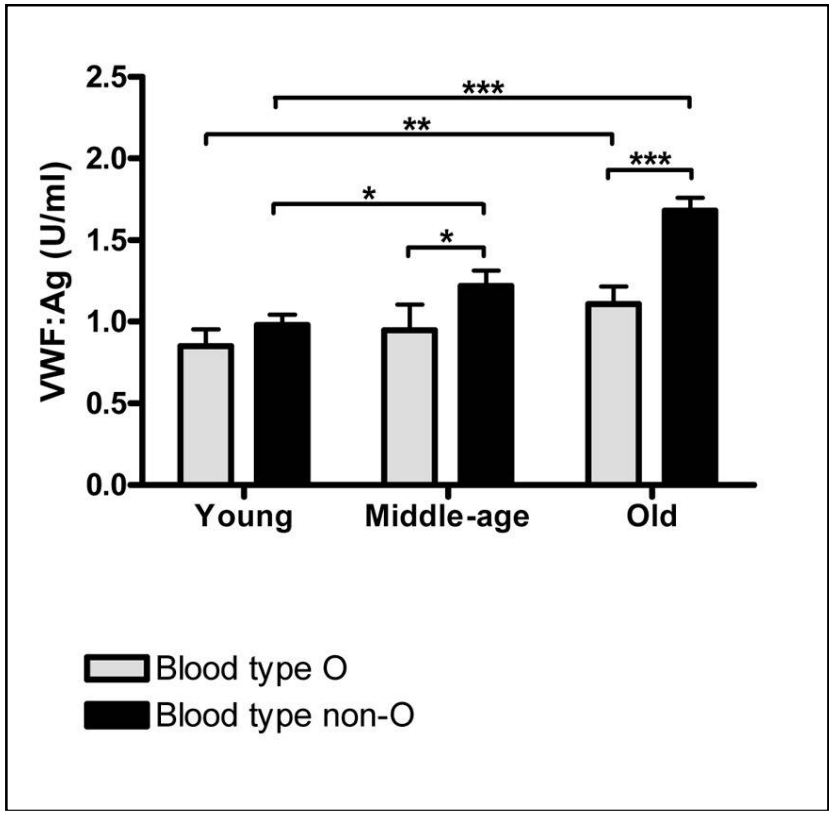
After 90 days of support, HM3 is associated with a 28.3% reduction in VWF HMWMs, compared with a 42.5% reduction in HMII



Bansal A et al. J Heart Lung Transplant. 2019

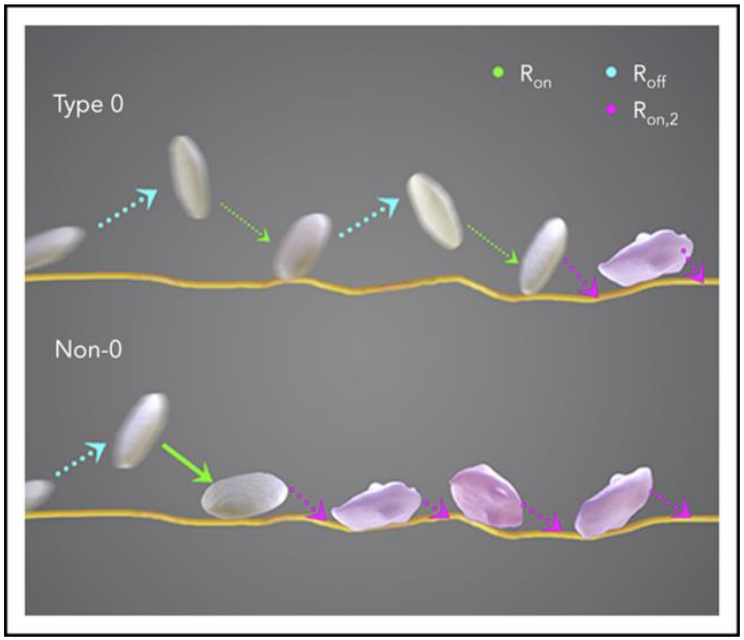
ABO blood groups influence vWF proteolysis

vWF-antigen levels are 25-35% lower in adults with blood type-O



Albánez S et al. J Thromb Haemost. 2016

Type O platelets travel **farther** and **stick less** on type O VWF



Dunne E et al. Blood. 2019

Objective

In the present exploratory analysis, we evaluated the association between Blood Type O and gastrointestinal bleeding following CF-LVAS implantation

Methods

Single center, retrospective, observational cohort study

Population: Consecutive adult patients with advanced heart failure who underwent surgical implantation of a durable CF-LVAS between May 2008 and March 2018

Exclusion: Patients with any paracorporeal, biventricular, or right ventricular assist systems, total artificial hearts, and CF-LVAS pump implanted as a 2nd pump for reasons of malfunction

Endpoint of interest: Gastrointestinal bleeding following the index hospitalization

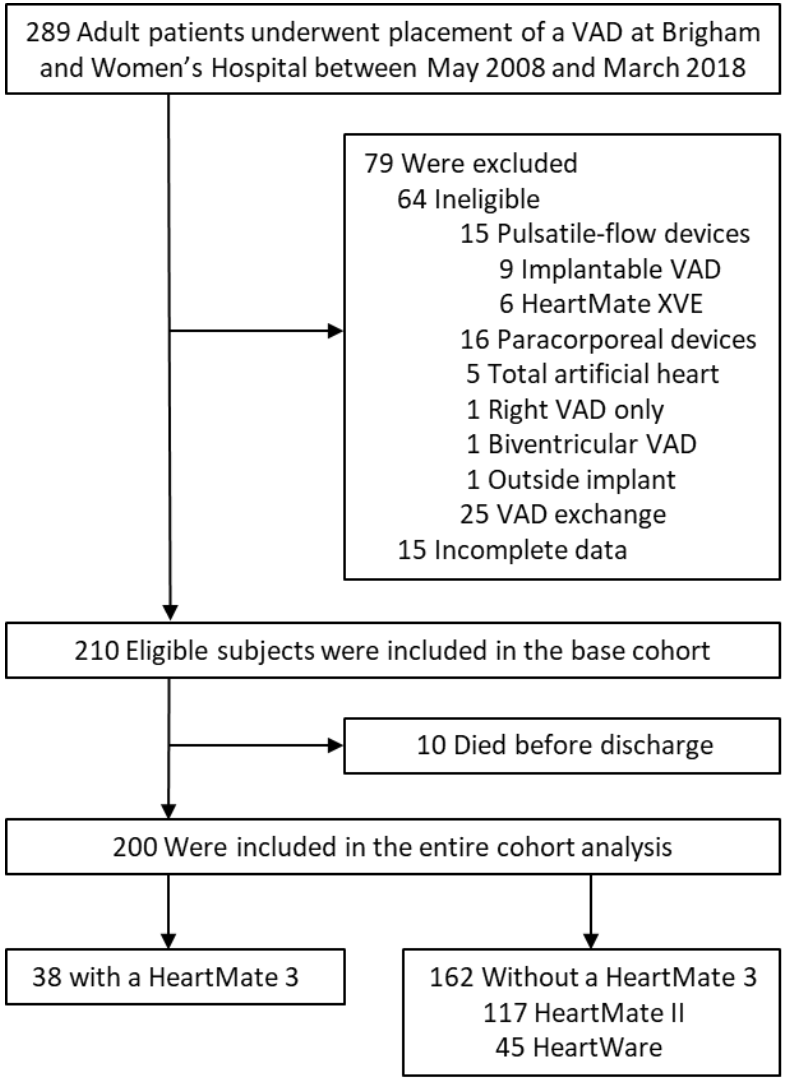
Risk of major bleeding was stratified according to the HAS-BLED score for an LVAS population

Koene RJ et al. J Card Fail. 2014
Kemal HS et al. ASAIO J. 2017

Eligible subjects were followed to end of study date (December 31, 2018) or until one of the following endpoints were reached: death, transplantation, CF-LVAS removal or replacement, or loss to follow up.

The study complies with the Declaration of Helsinki and was conducted with IRB approval

Flowchart of patients included in the study



Analysis

Baseline characteristics were compared according to Blood Type-O and Non-O

Categorical variables were compared with the $\chi 2$ or Fisher's Exact test

Continuous variables were compared with the Student's t-test or Mann–Whitney test

All **P values** are 2-tailed, and statistical significance set at $P < 0.05$

Confidence intervals were computed at the 95% confidence level

GIB was analyzed with the use of **Cox regression model**

Cumulative incidence curves were stratified by a HAS-BLED score of ≥ 3

Administrative **censoring** of data was performed for patients who were alive at the end of the study (December 31, 2018)

Statistical analyses were performed with **Stata software, version 15.0** (StataCorp)

Demographics

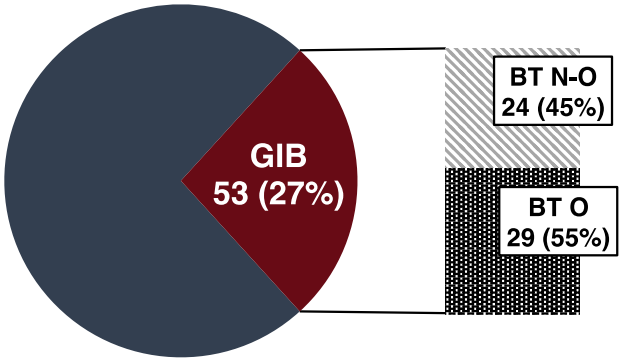
Variable	Overall Cohort N = 200	Blood Type O N = 93	Blood Type Non-O N = 107	P-value
Age, years	55 ± 13	55 ± 13	55 ± 14	0.84
Female, n (%)	37 (18.5%)	17 (18.3%)	20 (18.7%)	0.94
Race/Ethnicity, n (%)				
Caucasian	160 (80.0%)	73 (78.5%)	87 (81.3%)	0.62
Black/African American	15 (7.5 %)	7 (7.5 %)	8 (7.5 %)	0.99
Hispanic	8 (4.0 %)	3 (3.2 %)	5 (4.7 %)	0.60
Other/Unknow	17 (8.5 %)	10 (10.8%)	7 (6.5 %)	0.29
Hypertension, n (%)	109 (54.5%)	49 (52.7%)	60 (56.1%)	0.63
Diabetes Mellitus, n (%)	73 (36.5%)	33 (35.5%)	40 (37.4%)	0.78
History of Smoking, n (%)	93 (46.5%)	46 (49.5%)	47 (43.9%)	0.43
Ischemic Etiology, n (%)	75 (37.5%)	36 (38.7%)	39 (36.4%)	0.74
Body-Mass Index, kg/m^2	26.1 ± 5.6	26.5 ± 6.1	25.8 ± 5.1	0.35
Atrial Fibrillation, n (%)	83 (41.5%)	39 (41.9%)	44 (41.1%)	0.91
History of Stroke, n (%)	17 (8.5 %)	7 (7.5 %)	10 (9.3 %)	0.65
Chronic Kidney Disease, n (%)	60 (30.0%)	32 (34.4%)	28 (26.2%)	0.20
Peripheral Vascular Disease, n (%)	16 (8.0 %)	10 (10.8%)	6 (5.6 %)	0.18
Ejection Fraction < 20%, n (%)	127 (63.8%)	62 (67.4%)	65 (60.7%)	0.33

Demographics

Variable	Overall Cohort N = 200	Blood Type O N = 93	Blood Type Non-O N = 107	P-value
Type of CF-LVAS				
HeartMate II, n (%)	117 (58.5%)	59 (63.4%)	58 (54.2%)	0.19
HeartWare, n (%)	45 (22.5%)	18 (19.4%)	27 (25.2%)	0.32
HeartMate 3, n (%)	38 (19.0%)	16 (17.2%)	22 (20.6%)	0.55
Intended CF-LVAS Use				
Destination Therapy, n (%)	71 (35.5%)	32 (34.4%)	39 (36.4%)	0.76
Bridge Therapy, n (%)	129 (64.5%)	61 (65.6%)	68 (63.6%)	0.76
INTERMACS 1-2, n (%)	118 (59.0%)	51 (54.8%)	67 (62.6%)	0.26
Therapy at Implantation, n (%)				
Beta-blocker	144 (72.0%)	69 (74.2%)	75 (70.1%)	0.52
ACE inhibitor / ARB / ARNI	116 (58.0%)	55 (59.1%)	61 (57.0%)	0.76
Aldosterone antagonist	112 (56.0%)	53 (57.0%)	59 (55.1%)	0.79
Loop diuretic	184 (92.0%)	89 (95.7%)	95 (88.8%)	0.07
Statin	96 (48%)	46 (49.5%)	50 (46.7%)	
Inotrope therapy	160 (80.0%)	73 (78.5%)	87 (81.3%)	0.62
Laboratory at Implantation				
Creatinine, mg/dl	1.3 ± 0.5	1.3 ± 0.5	1.3 ± 0.5	0.69
Total Bilirubin, mg/dl	0.80 [0.50, 1.20]	0.80 [0.50, 1.20]	0.80 [0.50, 1.30]	0.29
AST, U/L	29 [21, 43]	28 [21, 47]	30 [21, 43]	0.58
ALT, U/L	30 [19, 47]	27 [19, 46]	31 [20, 47]	0.57
C-reactive protein, mg/dl	12.0 [5.1, 35.9]	12.2 [4.7, 34.0]	11.2 [5.1, 41.7]	0.59
LDH, U/L	288 [235, 404]	300 [242, 429]	280 [223, 385]	0.24
INR at Discharge	2.4 ± 0.5	2.5 ± 0.5	2.4 ± 0.4	0.29
Full-Dose Aspirin at Discharge, n (%)	134 (67.0%)	57 (61.3%)	77 (72.0%)	0.11
Blood Pressure at Discharge, mmHg	82.1 ± 9.5	83.3 ± 11.1	81.3 ± 8.3	0.31

Results

After a median follow-up of 10.4 months, 53 patients experienced at least one GIB event, of which 29 were BT-O

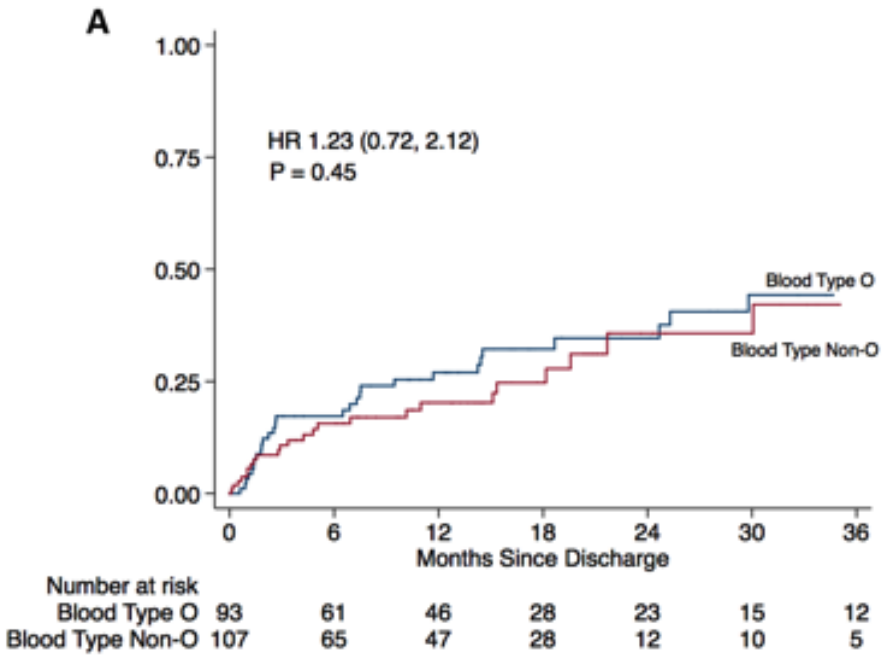


Eighty-seven (43.5%) patients had a **HAS-BLED score of ≥ 3**

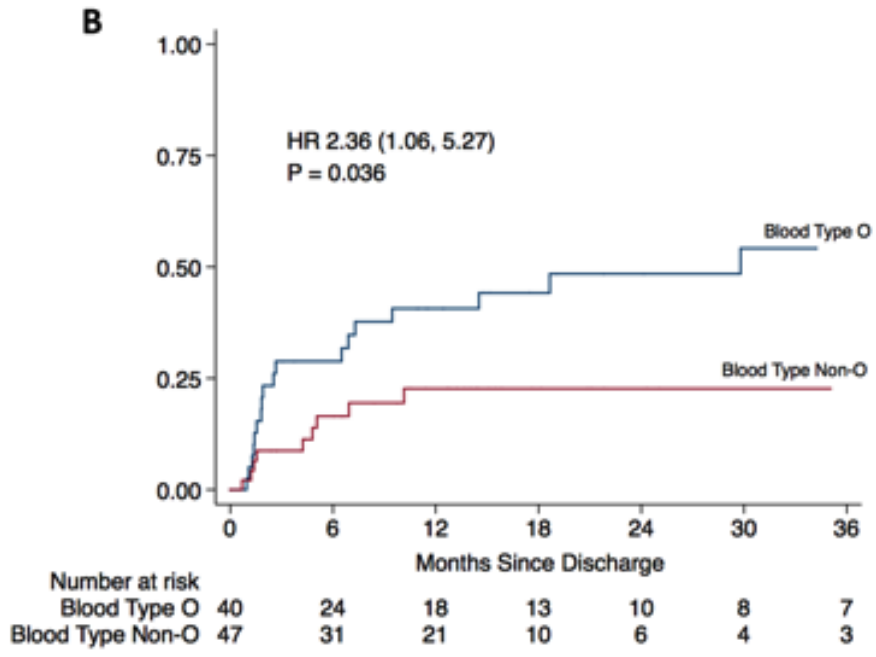
Results

Cumulative incidence of GIB following CF-LVAS implantation according to blood type

Overall cohort



Patients with HAS-BLED ≥ 3



Limitations

Observational study

Single-center

Limited generalizability

Conclusions

Our findings suggest that in this specific LVAS population with blood type O, a greater vigilance and individualized anticoagulation strategies may need to be investigated