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Managing Intracranial Hemorrhage in Patients with a Durable Continuous Flow Left Ventricular Assist Device

Godly Jack[1], Phil Barker[2], Ryan Searcy[2], and Jason N. Katz[3]

[1] Department of Internal Medicine, University of North Carolina School of Medicine, Chapel Hill, NC
[2] University of North Carolina School of Medicine, Chapel Hill, NC
[3] Division of Cardiology, University of North Carolina School of Medicine, Chapel Hill, NC

*Corresponding author: *katzj@med.unc.edu*

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Abstract

Background

While intracranial hemorrhage (ICH) is a known complication of left ventricular assist device (LVAD) support, and is associated with high morbidity and mortality, optimal care pathways have neither been elucidated nor reported. We describe management of LVAD patients following ICH, with a focus on anticoagulation, operative interventions, care team designation, complications, and outcomes.

Methods

We retrospectively reviewed all durable continuous-flow LVAD implantations at our academic medical center from January 2007 to July 2018. Patients who experienced ICH after LVAD were identified. We defined baseline and ICH characteristics, medical and surgical interventions, care teams, and outcomes including death, device thrombosis, ischemic stroke, and hemorrhage expansion.

Results

A total of 321 patients underwent LVAD implantation during the study period, and 27 (8%) developed ICH (17 intraparenchymal, 7 subdural, 2 subarachnoid, 1 intraventricular) while on support. Twenty-five were anticoagulated at onset of bleed. Of those, 13 were managed with immediate cessation of anticoagulation and administration of reversal products (Group A). Group A had a median of 6 days off anticoagulation and 60 days of follow up with 1 patient (8%) developing device thrombosis at day 8, 1 (8%) developing subsequent ischemic stroke at day 14, and 4 (31%) with ICH expansion. Seven patients had anticoagulation stopped at onset of bleed without administration of reversal products (Group B). With a median of 2 days off anticoagulation and 2 days of follow up, no patients in Group B developed ischemic stroke or device thrombosis while 1 (14%) had ICH expansion. Five patients had anticoagulation continued at onset of bleed (Group C) with a median follow up of 330 days. One (20%) developed device thrombosis at day 5 while 2 (40%) developed ICH expansion. Four patients with subdural hemorrhage underwent Burr hole drainage with all 4 surviving to discharge. Two patients with intraparenchymal hemorrhage underwent open craniotomy with neither surviving to discharge. An interdisciplinary discussion occurred in all cases. Following ICH, only one-third of patients in the study survived to 6 months.

Conclusion

LVAD patients who experience an ICH have variable outcomes. Their care is multidisciplinary and can involve operative intervention. The discontinuation and reversal of anticoagulation is generally well-tolerated, with a low risk for early device thrombosis. Like for many hemorrhagic complications of LVADs, ICH often persists or worsens. Additional investigation is needed to elucidate the most optimal management strategies.

Keywords: LVAD, intracranial hemorrhage, anticoagulation

Introduction

Though chronic heart failure is associated with substantial morbidity and mortality, the use of contemporary left ventricular assist devices (LVADs) has resulted in significant improvements among eligible patients (1-4). With that being said, there is still risk associated with device use. One well-recognized complication is that of bleeding, tied at least in part to the need for systemic anticoagulation. One potentially devastating bleeding event is that of intracranial hemorrhage (ICH). Those who develop ICH on LVAD support have markedly worse survival, and hemorrhagic strokes carry a considerably greater liability for LVAD patients than ischemic events (5, 6).

Although studies have identified risk factors associated with the development of ICH in LVAD patients, the actual management of this complication has been largely overlooked (7, 8). Treatment practices have been infrequently described and have been limited to small observational reports, and there are currently no guidelines for the management of ICH in this patient population. Additionally, the INTERMACS database, which collects clinical data including bleeding complications for LVAD patients, notably excludes details on the management of

ICH (9). For these reasons, treatment of ICH complicating LVAD use has been largely institution-dependent and driven by clinician experience and preference.

The current study was conducted in order to describe the management of LVAD patients following ICH, with a particular focus on anticoagulation, operative intervention, resource utilization, care team dynamics, complications, and clinical outcomes.

Methods

This study was reviewed and approved by the University of North Carolina (UNC) Institutional Review Board. Patients who received a continuous-flow, durable LVAD were identified using the UNC Mechanical Heart Program VAD tracking sheet which includes all patients at our institution who have received an intracorporeal mechanical circulatory support device since January 2007. We performed a thorough medical record review of those patients who underwent placement of a Jarvik 2000 (Jarvik Heart, Inc., New York, NY), HeartMate II (Thoratec, Pleasanton, CA), HeartMate III (Abbott, Chicago, IL) or HVAD (Medtronic, Dublin, Ireland) from January 2007 to July 2018 in order to identify those who had developed ICH following device implantation.

Data that were systematically abstracted from the electronic medical record included patient demographics (age, gender, and race) LVAD type, ICH type (IPH, intraparenchymal hemorrhage; SDH, subdural hemorrhage; SAH, subarachnoid hemorrhage; or IVH, intraventricular hemorrhage), days post-LVAD implantation, setting at ICH onset, mechanism of ICH, medical decision-making strategies (including management of antiplatelet and anticoagulation regimens, operative management including placement of Burr hole or open craniotomy), complications (including device thrombosis, ischemic stroke, and worsening bleed), resource consumption (including specialty care consultation and hospital unit placement), and mortality. Size of ICH was also calculated for IPH only (using the formula ABC divided by 2 where A = max height, B = max length, C = max depth). Location of bleed was collected for IPH and SAH (i.e. frontal, parietal, temporal, occipital, basal ganglia, or brainstem).

Those patients who were anticoagulated at the time of bleed were divided into three groups based on their anticoagulation management strategy: those who had anticoagulation immediately held with administration of reversal products (Group A), those who had anticoagulation immediately held without administration of reversal products (Group B), and those who had anticoagulation continued (Group C). Complication rates and survival outcomes were calculated for these three groups and stratified by type of hemorrhage.

Results

Study Population

From January 2007 to July 2018, 321 patients at our institution underwent placement of a durable, continuous-flow, intracorporeal LVAD, with 27 (8%) subsequently developing ICH while on mechanical support. The median time between LVAD implantation and ICH was 187 days (range = 4 – 1739 days). Among those who developed ICH, the average age at time of bleed was 63.1

years with the vast majority of patients being Caucasian or African American males with a HeartMate II (Table 1).

Table 1 – Patient characteristics, Clinical Data, and Management

N	Age, Sex, Race	LVAD	Presenting Symptom	ICH Type	ICH Size (cm^3)	INR	ICH Location	Days post-implant	Set-ting	Mech-anism	Anticoagulation Management	Surgery	Survival
1	58 WF	HMII	Altered Mental status	IPH	91	ADL	L temporal	158	OH	Sponta-neous	Warfarin held without reversal	None	<1 day
2	37 WM	Jarvik	Altered Mental Status	IPH	106	1.3	R frontal	75	IH	Sponta-neous	Heparin stopped without reversal	None	2 days
3	51 BM	HMII	Headache	SAH	N/A	3.3	R frontal	123	IH	Sponta-neous	Warfarin continued given history of APLA, DVT/PE, and LVAD thrombosis; switched to heparin day 4; warfarin restarted day 40 and heparin stopped 5 days after	None	> 6 months
4	74 WM	HMII	Headache	SDH	N/A	1.8	Right	266	OH	Traumatic	Warfarin held; given Vitamin K; warfarin restarted on day 30	None	> 6 months
5	64 BF	HVAD	Confusion	IPH	14	2.0	L temporal	105	IH	Traumatic	Warfarin plus heparin bridge stopped; given FFP and Vitamin K; warfarin restarted on day 4	None	> 6 months
6	62 WM	HMIII	Altered Mental Status	SAH	N/A	1.8	R parietal	143	IH	Sponta-neous	Heparin stopped; given protamine; warfarin and heparin bridge restarted on day 6	None	2 months
7	69 WM	HMII	Unrespons iveness	SDH	N/A	1.4	Right	198	OH	Traumatic	Warfarin held; given Factor 7	Cranio-tomy	< 1 day
8	68 WM	HMII	Unknown	SDH	N/A	1.4	L tentorium	329	IH	Traumatic	Not on anticoagulation given GI bleed 2 weeks prior; anticoagulation not restarted	None	49 days
9	58 NAF	HVAD	Unrespons iveness	IPH	unkn own	4.3	R fronto-parietal	86	IH	Sponta-neous	Warfarin held; given PCC, Vitamin K, and FFP	None	2 days
10	45 BM	HMII	Altered mental status, drowsi-ness	IPH	108	1.4	R occipito-parietal	122	IH	Sponta-neous	Heparin stopped; given protamine	None	< 1 day
11	65 BM	HMII	Headache, Nausea, vomiting	SDH	N/A	1.0	Bilateral	548	OH	Traumatic	Not on anticoagulation given GI bleed 2 weeks prior; warfarin restarted 30 days after ICH	Burr Hole Drain	> 6 months
12	69 WF	HMII	Nausea, Headache	IPH	105	2.9	L occipito-temporo-parietal	846	IH	Sponta-neous	Warfarin held without reversal	None	< 1 day
13	67 BM	HMII	Headache	IPH	27	5.1	R occipital	374	OH	Sponta-neous	Warfarin held; given FFP, PCC, Vitamin K, Factor 9, and Factor 2;	None	77 days

											warfarin restarted day 28		
14	79 BM	HMII	Headache, left arm symptoms	SDH	N/A	3.3	Right	701	OH	Sponta-neous	Warfarin held; given PCC and Vitamin K; warfarin restarted 64 days later	Burr Hole Drain	> 6 months
15	71 WM	HMII	Unknown	IPH	< 1	1.2	L frontal	4	OH	Hemorr-hagic Conver-sion	Heparin continued	Un-known	26 days
16	58 WM	HMII	Lethargy	IPH	26	1.59	L fronto-temporal	26	IH	Hemorr-hagic Conver-sion	Warfarin continued; heparin added as bridge for subtherapeutic INR	None	6 days
17	56 WM	Jarvik	Left-sided weakness	IPH	133	1.10	R fronto-parietal	616	IH	Sponta-neous	Warfarin held; given PCC and Vitamin K	None	2 days
18	72 WM	HMII	Incidental finding	SDH	N/A	2.3	Right	175	OH	Traumatic	Warfarin held without reversal and restarted on day 31	None	4 months
19	84 WM	HMII	Slurred Speech	IPH	16	3.58	L frontal	1739	OH	Hemorr-hagic Conver-sion	Warfarin held without reversal	None	< 1 day
20	43 BF	HMII	Slurred Speech	IPH	< 1	1.92	L frontal	160	OH	Hemorr-hagic Conver-sion	Warfarin continued	None	> 6 months
21	48 BM	HMII	Headache	SDH	N/A	3.51	Right	945	OH	Traumatic	Warfarin replaced with lovenox; warfarin restarted on day 3	Burr Hole Drain	> 6 months
22	55 BM	HMII	Headache, right hemipare-sis	IPH	10.8	1.7	L parietal	1220	IH	Sponta-neous	Heparin stopped; given FFP and Vitamin K; warfarin started on day 33	None	> 6 months
23	77 BM	HMII	Lethargy, right hemipares is	IPH	114.9 5	1.16	L frontal	99	OH	Sponta-neous	Warfarin held; given PCC and Vitamin K	Cranio-tomy	6 days
24	77 BM	HMII	Confusion Nausea	IVH	N/A	4.47	Right 3rd ventricle	1632	OH	Sponta-neous	Warfarin held; given PCC and Vitamin K; prophylactic heparin [(x)] day 3; ACS nomogram heparin[(y)] day 21; warfarin restarted day 47	Burr Hole Drain	> 6 months
25	64 WM	HMII	Left hemipares is	IPH	0.91	1.2	R basal ganglia	65	IH	Hemorr-hagic Conver-sion	Warfarin held without reversal, heparin drip on day 9	None	10 days
26	82 WM	HMIII	Altered Mental Status, Drowsi-ness	IPH	14.5	1.75	L occipital	29	IH	Hemorr-hagic Conver-sion	Warfarin and heparin both held without reversal	None	2 days
27	63 BF	HMII	Inattentive -ness, confusion	IPH	32	2.48	L frontal	149	OH	Sponta-neous	Warfarin held; given FFP and Vitamin K; heparin drip started day 8 for LVAD thrombosis	None	8 days

ADL = above detectable limit; BF=black female, BM=black male; HMII=HeartMate II; HMIII= HeartMate III; IH= in hospital; IPH=intraparenchymal hemorrhage; NAF= Native American Female; OH=out of hospital; PCC = prothrombin complex concentrate; SDH= subdural hematoma; WF=white female; WM=white male

(x) 5000 units unfractionated heparin every 8 hours (y) unfractionated heparin infusion titrated to goal heparin correlation of 0.3 – 0.5.

ICH Characteristics

Among the 27 cases of ICH, there were 17 intraparenchymal, 7 subdural, 2 subarachnoid, and 1 intraventricular event. Of the 17 patients who developed IPH, 10 presented with spontaneous hemorrhage, 6 with hemorrhagic conversion of an ischemic stroke, and 1 with head trauma. Of the 7 patients who suffered SDH, 6 had a history of head trauma or fall while 1 had no history of either. Of the 2 patients who developed SAH and 1 with IVH, none had a history of recent head trauma, fall, or ischemic stroke. Thirteen patients developed an ICH while inpatient, and of these 4 had been hospitalized for LVAD implantation, 4 were being treated for bacteremia or LVAD pocket infection, 2 for ischemic stroke, 1 for CHF exacerbation, 1 for pre-operative anti-coagulation management, and 1 for suspected LVAD thrombosis. Fourteen patients developed bleeding outside of the hospital (Table 1).

Anti-thrombotic Management & Complications

Twenty-one patients (78%) were on antiplatelet therapy at presentation with 19 (70%) on aspirin alone, one (4%) on prasugrel alone, and one (4%) on a combination of aspirin and clopidegrel. Only 4 patients had antiplatelet therapy continued at the time of bleed, all of whom were being treated for hemorrhagic conversion of an ischemic stroke. Three patients had antiplatelet therapy restarted at 1 week, 1 month, and 2 months respectively.

Twenty-five patients (93%) were anticoagulated at presentation with 19 (70%) on warfarin, 2 (7%) on a heparin infusion, and 4 (15%) on warfarin plus a heparin infusion. INR at time of presentation ranged from 1.1 to above the detectable limit. Due to recent history of GI bleeding, 2 patients (7%) were not anticoagulated at the time of their ICH. Among the 25 patients who were anticoagulated, 13 were managed with immediate cessation of anticoagulation and administration of reversal products (Group A), 7 were managed with immediate cessation of anticoagulation without administration of reversal products (Group B), and 5 were managed with continuation of anticoagulation (Group C). In Group A, reversal products utilized included Fresh Frozen Plasma (FFP), Prothrombin Complex Concentrate (PCC), vitamin K, and protamine. In Group B, many different clinical factors contributed to the decision to withhold reversal products. Specifically, 4 patients presented with unrecoverable bleeds and were managed with comfort measures only, 1 patient had an asymptomatic SDH for which reversal was deemed unnecessary, 1 patient had an ischemic stroke with a very small ICH from hemorrhagic conversion, and 1 patient had signs of chronic subclinical LVAD thrombosis. Given the poor prognosis of this group in general, their follow-up time was very limited. In Group C, anticoagulation was continued for several reasons.

Three patients had an ischemic or embolic stroke with two of the three having less than 1 cm^3 volume ICH, 1 patient had a SDH with intact neurologic status, and 1 patient had a history of antiphospholipid antibody syndrome complicated by prior LVAD thrombosis. In these 5 cases it was thought that the risk of discontinuing anticoagulation outweighed the benefits.

With a median follow-up of 26 days (range = 1 – 1350 days) in these 25 patients with ICH while on anticoagulation, there were a total of 2 patients with subsequent device thrombosis and 1 patient with ischemic stroke over the course of the study – none of which occurred within the first week after bleed. Group A had a median of 6 days off anticoagulation and 60 days median follow-up time with only 1 (8%) patient developing device thrombosis at day 8. One (8%) patient in this group developed ischemic stroke at day 14. Four (31%) had evidence of worsening bleeding which all occurred within 1 week (Figure 1). Group B had a median of 2 days off anticoagulation and 2 days of follow up time with no episodes of device thrombosis or ischemic stroke. One (14%) patient had evidence of worsening bleeding on day 1. Group C had a median follow-up time of 330 days with 1 (20%) device thrombosis at day 5 in a patient with a history of antiphospholipid antibody syndrome and multiple LVAD thromboses in the past. Expansion of ICH occurred in 1 (20%) patient on day 27 (Table 2).

Care Team Designation & Surgical Intervention

In all cases, an interdisciplinary discussion took place between cardiology, neurosurgery, neurology, cardiac surgery, and/or hematology. Eight (30%) patients had at least one consultant note that weighed the risk of device thrombosis heavily in clinical decision-making. Eighteen (67%) patients were treated in the cardiac intensive care unit (ICU), 2 (7%) in the neurosurgical ICU, and 6 (22%) in the cardiac intermediate unit (Table 3). One patient was taken to the operating room and died before being assigned to a hospital unit. Glasgow Coma Scale (GCS) was recorded only in 12 (44%) patients. NIH Stroke Scale (NIHSS) was recorded for 8 patients (30%) and Glasgow Outcome Scale (GOS) was not recorded for any patients.

Table 2: LVAD Thrombosis, Ischemic Stroke, Worsening Bleed, and Death Stratified by Anticoagulation Strategy

	Anticoagulation Management		
	Group A	Group B	Group C
	Reversed	Held	Continued
No. of patients	13	7	5
Follow up time (days), median	60	2	330
Days off AC, median[(a)]	6	2	-
Days off AC, range[(a)]	1 - 65	1 - 31	-
Device Thrombosis	1 (8)	0 (0)	1 (20)
Ischemic Stroke	1 (8)	0 (0)	0 (0)
Expansion of ICH [(b)]	4 (31)	1 (14)	1 (20)
Death at 24 hours (%)	2 (15)	3 (43)	0 (0)
Death at 30 days (%)	6 (46)	6 (86)	2 (40)
Death at 6 months (%)	8 (62)	7 (100)	2 (40)

(a) Ended with either resumption of anticoagulation or death

(b) defined as clinical or radiographic evidence of hemorrhage expansion

AC = Anticoagulation

ICH= Intracranial hemorrhage

Table 3: Care Team and Hospital Unit

No. of Patients, total [(a)]	27
Primary Team (%)	
Cardiology	21 (78)
Cardiac Surgery	2 (7)
Neurosurgery	3 (11)
Consultants (%) [(b)]	
Neurosurgery	20 (74)
Neurology	14 (52)
Cardiology	5 (19)
Hematology	2 (7)
Hospital unit [(a)]*(%)*	
CICU	18 (67)
NSICU	2 (7)
Cardiac Intermediate Care	6 (22)

(a) One patient was taken to the OR and died before being assigned to a unit and before consult teams could be assigned

Surgical intervention was performed in a total of 6 patients. This included 4 patients with SDH who underwent Burr hole drainage and 2 patients with IPH who underwent open craniotomy.

Outcomes

Survival outcomes varied by management group (A, B, and C) and by ICH subtype (Table 2 and 4, respectively). Thirty-day mortality was 46%, 86%, and 40% in Groups A, B, and C respectively, while 6-month mortality was 62%, 100%, and 40% respectively. Patients who presented with IPH had the worst outcomes with a 76% rate of death at 30 days compared to only 14%, 0%, and 0% in patients with SDH, SAH, and IVH respectively. Both patients who underwent open craniotomy for IPH died within 6 days. On the other hand, all four patients who underwent Burr hole drainage survived to 6 months.

Table 4: Survival Outcomes

	IPH	SDH	SAH	IVH	All
Death at 24 hours (%)	4 (24)	1 (14)	0 (0)	0 (0)	5 (19)
Death at 30 days (%)	13 (76)	1 (14)	0 (0)	0 (0)	14 (52)
Death at 6 months (%)	14 (82)	3 (42)	1 (50)	0 (0)	18 (72)

IPH= intraparenchymal hemorrhage; IVH= intraventricular hemorrhage; SAH= subarachnoid hemorrhage; SDH=subdural hemorrhage

Discussion

In this case series assessing anticoagulation management, surgical intervention, care team utilization, complications, and outcomes among continuous-flow LVAD patients presenting with ICH, we found that the majority of patients were middle-aged males with a Heartmate II device, almost all of whom were anticoagulated at presentation. The etiology of ICH included traumatic, spontaneous, and hemorrhagic conversion with about half of patients developing ICH while already in the hospital for an alternative reason. Clinical management strategies varied across our patient population with mixed outcomes. Most notably, cessation of anticoagulation with administration of reversal products was generally well tolerated in the short term, with no episodes of device thrombosis or ischemic stroke occurring within the first week when this strategy was employed. On the other hand, about one-quarter of patients in the study had clinical or radiographic evidence of worsening bleeding; the majority of which did occur within the first week. Intracranial hemorrhage was associated with poor outcomes as only one-third of patients in the study survived to 6 months.

While the management of anticoagulation in the LVAD patient with ICH remains a unique and challenging dilemma, the data currently available to guide treatment remains limited. In a single-center study of LVAD patients presenting with ICH, Wilson et al followed 22 individuals who had warfarin held and reversed for a median of 10.5 days and found that none developed device thrombosis or stroke (10). This study, however, was performed largely among patients supported with a pulsatile LVAD rather than a contemporary continuous-flow device. A similar study by Wong et al analyzed the efficacy of vitamin K antagonist reversal using 4-factor PCC versus traditional reversal strategies in continuous flow devices and found only one LVAD thrombosis and no ischemic strokes among 20 cases (11). This study was also limited by the fact that the average time off anticoagulation was not reported, making it difficult to interpret and generalize. In the largest observational study to date, Tahir et al found that ICH size, midline shift, and GCS score were predictive of mortality, but they did not report data on reversal strategies or rates of device thrombosis (12). Our study revealed no device thrombosis or ischemic stroke within the first week in patients managed with swift cessation and reversal of anticoagulation. However, almost one-third of patients had consult notes which weighed the risk of device thrombosis and ischemic stroke heavily in clinical decision-making. Additionally, a significant proportion of patients across all treatment groups demonstrated clinical or radiographic evidence of worsening

bleeding within the first week. One could therefore make a compelling argument that in the short term, the risk of bleeding may outweigh the risk of clotting in such cases. With the fully magnetically-levitated HeartMate 3 demonstrating lower rates of ischemic stroke and pump thrombosis but similar rates of bleeding compared to the older HeartMate 2, we expect this pattern may persist (13, 14).

The role of neurosurgical intervention in this patient population remains even more unclear. Although small studies suggest non-cardiac surgery in the LVAD patient does not significantly increase morbidity and mortality, very few specifically assess outcomes after intracranial hematoma evacuation (15-17). In a recent study by Ikeda et al, 6 of 7 LVAD patients with ICH who underwent acute operative intervention (6 craniotomies and 1 burr hole drainage) died within 30 days (18). Wilson et al reported similar results, with four of five patients who underwent craniotomy dying within 60 days (10). At our institution, both patients treated with open craniotomy died within 1 week while all 4 patients who underwent Burr hole drainage for subdural hematoma survived to 6 months. Other studies have also shown Burr hole drainage to be generally well tolerated in these cases (10). Whether the poor outcomes seen with craniotomy are due to sicker patients going to the OR is unclear, and more data is needed to make definitive recommendations. Notably, the 2015 American Heart Association/American Stroke Association guidelines on the management of spontaneous intracranial hemorrhage state that, in general, the role of surgery for supratentorial ICH is not well established (19).

Our study is the first to describe care team designation and hospital unit admission patterns as key factors that may impact patient management. The majority of patients were treated in either a cardiac ICU or a cardiac intermediate care unit with cardiac intensivists or cardiac surgeons serving as leaders of the care team. Two patients were managed in the neurosurgical ICU. With very few exceptions, all patients were also followed by a neurosurgery and/or neurology consultant team. Whether to manage these patients in the cardiac versus neurosurgical ICU remains controversial, with clinical resources and specialized nurse training playing a key role in this debate. The cardiac ICU allows for closer and more experienced monitoring of potential LVAD dysfunction such as device thrombosis, arrhythmia, and heart failure, whereas the neurosurgical ICU may allow for more optimal management of ICH including monitoring of intracranial pressures, tracking of cerebral perfusion pressures, early identification of dynamic changes in neurologic function, and other tasks that may benefit from more specialized nursing care (20). As an example, we found that less than half of our patients who were treated in a cardiac unit had either Glasgow Coma Scale (GCS) or NIH Stroke Scale (NIHSS) recorded during their hospital stay, and none of the patients had a Glasgow Outcome Scale documented. Given that GCS and other stroke assessment scores have been shown to be important in the management of ICH, and that the AHA/ASA recommends that nurses caring for these patients be trained in obtaining these scores, this study highlights the need to more closely examine these care practices in our cardiac-specific ICUs (19). Given the low risk of early device thrombosis in our patient population, the potential for rapid neurologic decline, and the level of specialized care needed to treat an acute intracranial bleed, one might consider treating LVAD patients with ICH in a neurosurgical ICU or dedicated stroke unit rather than the cardiac ICU. The risks

and benefits of this approach should currently be evaluated on a case-by-case basis, but we believe this strategy could and should be more rigorously assessed in a prospective clinical study.

In addition to prospective clinical assessment, we suggest that more data elements concerning ICH be collected as part of the INTERMACS database. This should include ICH subtypes, management practices (including pharmacologic reversal agent use and operative interventions), and neuro-specific outcomes. More data is certainly needed to understand which components of management truly influence patient outcomes.

Our study utilized rigorous chart review to gather clinical data not previously reported in larger studies, and is the first to collect data on care team designation and hospital unit following ICH. There are, however, several limitations that should be considered. First, given the small sample size our results should be viewed as largely descriptive. We did not have the necessary power to make direct statistical comparisons across groups. Instead we focused on providing rigorous data related to care processes and stroke characteristics that we hope will serve as a basis for future investigation. Second, the majority of patients in Group B suffered devastating bleeds, were transitioned to comfort measures within 1 or 2 days of admission, and died shortly thereafter, thus limiting their follow up time. Finally, as previously mentioned, our records did not consistently include data relevant to stroke outcomes such as GCS, NIHSS, GOS, and Rankin scale.

Conclusion

Patients with LVADs who experience an ICH have very high mortality rates. Their care is multidisciplinary and can involve operative intervention in certain circumstances. Early reversal of anticoagulation is generally well tolerated with a low risk of early device thrombosis or ischemic stroke. Like for many hemorrhagic complications of LVADs, bleeding after ICH often persists or worsens. Additional prospective studies are needed to provide future guidance to clinicians faced with this difficult, and often devastating clinical event.

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