The Use of Novel Oral Anti-Coagulant's (NOAC) compared to Vitamin K Antagonists (Warfarin) in patients with Left Ventricular thrombus after Acute Myocardial Infarction (AMI)

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Abstract

Aim: Current guidelines recommend the use of Vitamin K Antagonist (VKA) for up to 3 – 6 months for treatment of LV thrombus post-acute myocardial infarction (AMI). However, based on evidence supporting non-inferiority of Novel Oral Anti-Coagulant's (NOAC) compared to VKA for other indications such as DVT, PE and thrombo-embolic prevention in atrial fibrillation, NOACs are being increasingly used off licence for the treatment of LV thrombus post AMI. In this study we investigated the safety and effect of NOACs compared to VKA on LV thrombus resolution in patients presenting with AMI.

Methods and Results: This was an observational study of 2,328 consecutive patients undergoing Coronary Angiography +/- Percutaneous Coronary Intervention (PCI) for AMI between May 2015- December 2018, at a UK cardiac centre. Patients' details were collected from the hospital electronic database. The primary end-point was rate of LV thrombus resolution with bleeding rates a secondary outcome.

Left ventricular (LV) thrombus was diagnosed in 101 (4.3%) patients. Sixty patients (59.4%) were started on VKA and 41 patients (40.6%) on NOAC therapy (rivaroxaban: 58.5%, apixaban, 36.5% and edoxaban: 5.0%). Both groups were well matched in terms of baseline characteristics including age, previous cardiac history (Previous MI, PCI, CABG), and cardiovascular risk factors (Hypertension, Diabetes, Hypercholesterolaemia).

Over the follow up period (median 2.2 years), overall rates of LV thrombus resolution were 86.1%. There was greater and earlier LV thrombus resolution in the NOAC group compared to patients treated with warfarin (82% vs 64.4%, p=0.0018, at 1 year), which persisted after adjusting for baseline variables (OR 1.8 95% CI 1.2-2.9). Major bleeding events during the f/u period were lower in the NOAC group, compared with VKA group (0% vs 6.7%, p=0.030) with no difference in rates of systemic thromboembolism (5% vs 2.4%, p=0.388).

Conclusion: This data suggests improved thrombus resolution in post ACS LV thrombosis in patients treated with NOACs compared to vitamin K antagonists. This improvement in thrombus resolution was accompanied with a better safety profile for NOAC patients' vs VKA treated patients. Thus, provides data to support a randomised trial to answer this question.

Introduction

Left ventricular (LV) thrombus is a recognised complication of acute myocardial infarction (AMI) and is associated with a significant thromboembolic risk when left untreated (1, 2). Despite advances in interventional cardiology with timely and effective mechanical reperfusion, the incidence of LV thrombus in AMI, particularly in anterior AMI patients, remains, with reported incidences between 2.7% - 12.3% of all cases (2, 3, 4, 5), with a higher incidence of thrombi (between 11.5% and 43%) occurring in those with anterior ST-elevation myocardial infarction (STEMI) (6). Due to a reported incidence of 10-40% of thromboembolic events occurring in these patients current guidelines (ESC, ACCF/AHA, and ACCP) recommend the use of a Vitamin K Antagonist (VKA) for up to 3 – 6 months in patients with confirmed LV thrombus post-AMI (7) (8) (9).

Novel oral anticoagulants (NOACs) are highly selective direct factor Xa or thrombin inhibitors with oral bioavailability and rapid onset of action (10). They have predictable pharmacokinetics across a wide spectrum of patients (age, gender, weight) and have been found to be non-inferior or even superior compared to warfarin in the prevention of thromboembolic events in patients with non-valvular atrial fibrillation, treatment of thrombo-embolism (e.g. pulmonary embolism or deep vein thrombosis (DVT)), and prevention of DVT (11) (12) (13). NOACs are therefore increasingly used for off licence indications such as the treatment of LV thrombus, due to their fixed dosing regimes, low rate of drug interactions, and patient convenience with no need for frequent blood tests and possible long-term cost savings. However, the assumption of comparability between NOACs and warfarin for other indications is potentially problematic for example, higher rates of both ischaemic stroke and bleeding were seen when dabigatran was used for thromboprophylaxis of mechanical heart valves (14), and a recent observational study suggested superiority for warfarin in all comers with LV thrombus (15).

In this study we investigated the contemporary incidence of LV thrombus in AMI patients in the primary PCI era, looking at predictors and outcomes specifically assessing the effect of NOAC and VKA (warfarin) on LV thrombus resolution. We also assessed resolution time, rates of major bleed, and systemic thromboembolism, comparing VKA and NOAC use.

Methods

Study design and patient population: This was a single-centre, cohort study of 2,328 patients conducted at Barts Heart Centre from May 2015 to December 2018. This study was designed to assess incidence and outcomes of LV Thrombus but specifically the use of NOAC's as treatment for LV thrombus in patients presenting with AMI undergoing primary PCI, providing supportive data for further randomised study. Patients presenting with Non-ST elevation MI, treated for LV thrombus secondary to alternative aetiologies or already anti-coagulated for other reasons e.g. AF were not included in the analysis.

Assessment of left ventricular thrombus: In this study, LV thrombus was assessed using transthoracic echocardiography, or cardiac magnetic resonance imaging (CMR). All patients underwent a screening echocardiographic examination on admission and full study 12–24 h after admission. With transthoracic echocardiography, LV thrombus was defined as an echodense mass adjacent to an abnormally contracting myocardial segment. LV thrombus had to be distinguishable from the underlying myocardium, have a clear thrombus–blood interface, and be visible in more than two views. Echocardiography findings were interpreted independently by two expert cardiologists, with contrast studies performed as per departmental policy in uncertain cases. With gadolinium-enhanced CMR, LV thrombus was identified as a LV mass with low-signal intensity surrounded by high-signal intensity structures such as intra-cavitary blood or hyper-enhanced infarcted myocardium. Left ventricular thrombus was carefully distinguished from microvascular obstruction, which also appears as a dark area in the site of infarction, based on the established criteria. Cardiac magnetic resonance imaging was performed according to the discretion of the attending physicians.

Treatment: Patients with confirmed LV thrombus were anti-coagulated during their in-hospital stay (range 2-6 days post presentation) with either a Vitamin K Antagonist (Warfarin) or NOAC (apixaban, edoxaban or rivaroxaban). Patients not presenting with acute or recent AMI were not included in this study. Choice of VKA or NOAC was according to the discretion of the attending physicians, with low molecular weight heparin (LMWH) used until diagnosis confirmed. Off label use of NOAC was discussed/offered to patients with a confirmed diagnosis of LV thrombus. NOAC dose was decided based upon guideline recommendations and patient specific factors (i.e renal failure, bleeding risk). Patients with LV thrombus were counselled about the guideline-endorsed use of warfarin as the preferred first line of treatment with the limited data for NOAC in the treatment of LV thrombus

management discussed. This was explained by the physician and a pharmacist. For those patients selecting NOAC, the prescription was promptly filled, with the first dose administered the same day. Patients initiated on warfarin were treated with concomitant LMWH until warfarin was therapeutic. We targeted a therapeutic prothrombin time using an international normalised ratio (INR) range of 2.0-3.0 according to ESC guidelines. Triple therapy was defined as the combination of dual antiplatelet therapy (DAPT) plus an oral anticoagulant. Aspirin and clopidogrel was the preferred choice of DAPT if deemed necessary with anticoagulation, as per ESC guidelines, no potent P2Y12 inhibitors (ticagrelor or prasugrel) were used in combination with oral anti-coagulation.

Data Collection: Data were prospectively entered into a database at the time of the procedure, with PCI data entered in accordance to the British Cardiovascular Intervention Society (BCIS) standards. Data collected included patient characteristics (age, prior myocardial infarction (MI), PCI and coronary artery bypass grafting (CABG), hypertension, diabetes mellitus, hypercholesterolemia, smoking status, and cardiogenic shock) and procedure related data (indications for PCI, target vessel, number of diseased vessels, use of intravascular ultrasound (IVUS), optical coherence tomography (OCT), pressure wire, use of drug-eluting stent and GPIIb/IIIa inhibitor).

Endpoints: The primary aim was to assess the resolution of LV thrombus measured over the study follow-up period (median 2.2 years). All patients underwent imaging (TTE or CMR) as planned by the treating physician. Secondary endpoints included bleeding events (defined by BARC criteria (16)), thromboembolic events, and death and were recorded prospectively. These events were adjudicated by 3 independent physicians [DJ, AB, DW] who were not involved in the procedure and were unaware of the patient's treatment, using the patient's electronic hospital records. Procedural complications recorded included MI, emergency CABG, arterial complications, aortic/coronary dissection/perforation, side branch occlusion, and arrhythmia. Procedural complications were recorded at the time of the procedure and in hospital complications were entered into the database at the time of discharge.

Outpatient INR values measured during months 1–6 after the initial prescription of VKA were used to classify patients as having 'good control' (time in target range (TTR) $\geq$ 65%) or suboptimal control (TTR <65%) (17). We calculated TTR using the method of Rosendaal, which incorporates both the frequency of INR measurement and the actual values to interpolate daily INR values and define the percentage of time in range for each patient (18).

Ethics: Data were collected as part of a national cardiac audit, with the off-license use of NOACs discussed with each patient at the time. The local ethics committees advised that formal approval was not required.

Statistical Analysis: Baseline patient, procedural, and post-procedural characteristics were compared between the 2 groups. Categorical data are summarized using absolute values (percentage). Normally distributed, continuous data are presented as mean±SD or, where skewed, as median (25^{th} to 75^{th} centile). Normally distributed continuous variables were compared using Student t tests, and the Mann-Whitney U test was used to compare non-normally distributed continuous variables. Categorical data were compared using the Pearson chi-squared test. Multivariable logistic regression analysis was used to determine independent predictors of LV thrombus formation, using selected covariates. Variables associated at univariate analysis with LV thrombus (all with $P<0.05$) and those judged to be of clinical importance from previously published literature were eligible for inclusion into the multivariable model. Model discrimination was measured by the C-statistic and calibration by the Hosmer-Lemeshow goodness-of-fit test. The Cochran–Armitage test for trend was used to test the changes over time. Long-term survival was described by the Kaplan-Meier method and comparisons in LV thrombus resolution and survival between groups were made using the log-rank statistic. Cox regression analysis was used to estimate hazard ratios in the entire population and fully adjusted models, based on covariates ($p<0.05$) associated with the outcome. Variables included in the model including age, gender, diabetes mellitus, hypertension, hypercholesterolaemia, previous CABG, previous PCI, previous MI, previous cerebrovascular accident (CVA), peripheral vascular disease (PVD), multi-vessel disease, Chronic Renal Failure (CRF), left ventricular ejection fraction (LVEF), coronary artery treated, and PCI or medical treatment. A two-sided $P<0.05$ was considered statistically significant. All statistical analyses were performed using SPSS version 21.0 (*SPSS Inc*).

Results

A total of 101 (4.3%) patients with a mean age 59.61 +/- 14.08 years were identified to have LV thrombus. Of these acute STEMI patients, the majority (87.1%) presented with anterior infarcts (88 patients). In terms of left ventricular ejection fraction (LVEF), 77 (76.2%) had a LVEF <40% with a mean LVEF of 34.48% +/-15.0 for the study cohort. 21 patients (20.7%) did not undergo early catheter-based revascularization therapy, with the most frequent reason late presentation at the hospital (>24 h from AMI onset) in 16 (76.2%) of these patients. In the remaining patients, intervention was not performed because of myocardial infarction related to previous coronary artery bypass grafting and an undetermined culprit lesion.

Patients with LV thrombus tended to be younger, male and present with anterior MI (P<0.0001), higher peak troponin T and lower LVEF than patients without LV thrombus (Table 1). Of the 101 patients with confirmed LV thrombus, 60 patients (59.4%) were started on warfarin and 41 patients (40.6%) were treated with NOAC therapy. The most commonly used NOAC was rivaroxaban in 58.5% of cases (15mg in 46.2%, 20mg in 12.3%) followed by apixaban in 36.5% (2.5mg bd in 17%, 5mg bd in 19.5%) and edoxaban in the remaining 5% (30mg in 2.5% and 60mg in 2.5%) of cases.

For diagnosis of LV thrombus all patients underwent screening echocardiograms post PCI (+/- Contrast in 22.8%) to identify thrombus. 70.3%of these patients also underwent additional CMR pre-discharge with no difference seen in CMR utilisation for diagnosis between groups (66.7% VKA, 73.2% NOAC, P=0.518)

Baseline patient, presentation and procedural characteristics (Table 2)

Both groups were well matched in terms of baseline characteristics including age, previous cardiovascular history, and cardiovascular risk factors. The degree of LV impairment (LVEF < 40%) was similar in both groups, with similar usage of DES. Similar rates of patients not undergoing revascularisation and use of DAPT at discharge were seen between the groups. Among all patients with LV thrombus, 70 patients (69.3%) were on triple therapy at discharge with a median duration of 3 months (IQR 1-3 months). No difference in the rate of concomitant DAPT was seen between groups (70.0% VKA vs 68.3% NOAC) or duration (1 month: 19.0% VKA, 21.4% NOAC, 3 months: 81% VKA, 78.6% NOAC).

LV Thrombus Resolution

Sixty-five (64.3%) of the 101 patients underwent a follow-up CMR with 36 (35.7%) undergoing TTE surveillance. No difference in the utilisation rates of CMR was seen between the two groups (61.7% CMR in VKA, 68.3% NOAC (p=0.532)). Overall rates of LV thrombus resolution were 86.1% over the follow-up up period with an average median time to thrombus resolution of 211 days. Median time to 1st imaging was 151 days (IQR 63-352) for the NOAC group and 175 days (98-340) for the VKA group (P=0.414). 29 patients (70.7%) in the NOAC group and 29 patients (48.3%) in the VKA had resolution of thrombus noted on their first follow-up imaging (p=0.04). Over the remaining time period there was a consistent significant improvement in LV thrombus resolution with NOAC use compared to VKA use (p=0.002). Comparing treatment outcome at different time points (figure 1) throughout the follow up period demonstrated quicker thrombus resolution in the NOAC treated group compared to VKA group (1-year 82% vs 64.4%, p=0.0018). This difference in resolution rate persisted after adjusting for baseline variables (OR 1.8 95% CI 1.2-2.9). Thrombus resolution rates were 88%, 93%, and 100% for rivaroxaban, apixaban, and edoxaban, respectively. Post discontinuation of OAC, 38 patients (43.6%) underwent further surveillance imaging (81.5% TTE) which showed no recurrence of thrombus.

Oral anticoagulation was discontinued in 76 (75.2%) of 101 patients during the follow-up period. The most common reasons for anticoagulation discontinuation were disappearance of LV thrombus in 71 (93.4%) patients, a bleeding event in 4 (5.3%) patients, and patient preference for one patient. Overall median duration of treatment with NOACs was significantly shorter (5.1 months vs 8.7months) compared to warfarin therapy (p=0.02), due to earlier thrombus resolution.

Time in Range

Overall of the patients taking VKA's, 32 patients (53.3%) were classified as having good control with the remaining 28 (46.7%) suboptimal. Of those with suboptimal control, 22 (75%) were below the therapeutic range with 6 (25%) above the therapeutic range. A trend to greater thrombus resolution was seen in patients who had good compared to sub-optimal control over the follow-up period (p=0.0822).

Thromboembolism

Overall information on secondary endpoints of thromboembolism and bleeding were available in all patients with none lost to follow up (Median length of f/u 2.2years). Rates of systemic thromboembolism were low in the study cohort (4.0%) with all events involving the brain (n=4, 100%). No difference in event rates were seen between the 2 treatment groups (NOAC 2.4% vs VKA 5%, p=0.388) over the follow-up period. All the event rates in the VKA

group occurred in individuals who had sub-optimal TTR (<65%), with no events occurring in those with good control. No events occurred in patients on triple therapy, with events occurring in patients on single (n=1, VKA group 8 months) or no anti-platelet agents (n=3) (VKA group, events at 9 and 12 months, NOAC group event at 13 months).

Bleeding

Similar bleeding risks as assessed by the HAS-BLED score were documented in the 2 groups with no difference seen between the 2 groups. The incidence of major bleeding events as assessed by (BARC >2) in the VKA group was 6.7% compared to the NOAC group (0%, p=0.03). These events included gastrointestinal bleeding (3.3%), sub arachnoid haemorrhage (1.7%), and epistaxis requiring hospital admission (1.7%). These bleeding events occurred on triple therapy in 2 patients and dual therapy in the other 2. No major bleeding events were seen with adjunctive anti-platelet therapy in the NOAC group. Minor bleeding events (BARC 1) were numerically but not statistically smaller in the NOAC group (0.318).

Discussion

The major findings of this study are: (i) the prevalence of LV thrombus was 4.3% in patients presenting with acute myocardial infarction in our centre; (ii) overall rates of thrombus resolution were 86.1% during the follow up period (iii) there were greater and earlier resolution of LV thrombus in the NOAC group compared to patients treated with warfarin (81% vs 63%, p=0.0018, at 1 year), which persisted after adjusting for baseline variables (OR 1.8 95% CI 1.2-2.9); (iv) major bleeding such as as intracranial bleed, major GI bleed and bleed requiring hospital admission were lower in NOAC group, compared with VKA group (0% vs 6.7%, p= 0.030) with no difference in rates of systemic thromboembolism (p=0.388).

Previous studies have reported the incidence of LV thrombus in patients presenting with AMI ranging from 2.7% to 12.3% (19) (20) (21) (2, 4, 5)**,** with a higher incidence of thrombi (between 11.5% and 43%) occurring in those with anterior STEMI (6) (22) The contemporary incidence of LV thrombus (4.3%) in this study is likely to represent the improvement that has been seen in re-perfusion therapy with primary PCI and the use of echocardiography as the first line cardiac imaging for primary screening of LV thrombus. Using delayed enhancement CMR as the reference, echocardiography is shown to have a sensitivity of 33%, specificity of 91% and positive and negative predictive values of 29% and 93% respectively (23) (24). CMR was used to confirm equivocal cases and monitor resolution rather than being the screening tool, which may have led to higher rates of LV thrombus detection.

Current guidelines recommend the use of VKA in the treatment of LV thrombus. There are clinical limitations in its use; in particular multiple food and drug interaction, slow onset of actions, narrow therapeutic range, haemorrhagic complications, the need for frequent monitoring and therefore long-term cost implications. These limitations can be overcome by the use of NOACs as they have shown to have predictable efficacy, fixed dosing strategy, favourable safety profile, fewer drug interactions, and more rapid onset of effect, consistent anticoagulation and without the need for frequent blood monitoring. In addition, a potential benefit of NOACs over VKA is the statistically significant reduction in the risk of intracranial haemorrhage (ICH) (~50% RRR) (25), although less evidence is available to support this reduction with the addition of single or dual anti-platelet therapy. Consistent ICH rates of 1-1.3% are seen with VKA and DAPT (triple therapy) in registries (26), or large RCTs (27–29), which is similar to the rates seen in our study. Despite.the hazard ratio of ICH being reduced when using a NOAC in place of warfarin in a Danish registry (patients over >50 years with

AF) from 1.61 (1.23-2.10) in comparison to using VKA monotherapy vs a hazard ratio of DOAC triple therapy of 0.9 (0.34-2.40) (30), similar significant results have not yet been replicated in randomised studies nor in this analysis despite promising trends (28, 29). Noting the high incidence of triple therapy in our population, recommendations of aspirin and clopidogrel in combination of a NOAC could suggest a reduction in major bleeding overall and in particular the risk of ICH, although further study is still needed.

Previous case studies have demonstrated NOACs to be effective in patients with LV thrombus and AMI, with case studies showing efficacy of rivaroxaban (31), apixaban (32), dabigatran (110mg twice daily) (33) and edoxaban (34), with a review of all case series suggesting safety of the drugs (35). However, despite these positive case studies a recent observational study of 514 patients with LV thrombi (all indications) showed a significant association between NOAC use and systemic embolization when compared to warfarin use (15). Importantly though this detrimental signal was lost when just patients with an ischaemic aetiology of LV thrombus i.e. AMI or cardiomyopathy were looked at suggesting possible equivalence or non-inferiority in this group. Although there are no current randomised controlled trials or guideline recommendations considering the use of NOACs for treatment of LV thrombus in AMI setting, findings of our study are encouraging and support its off-label use for such purposes. As data on their use in the treatment of LV thrombus are limited to small number of case studies and observational studies, randomised controlled trials are needed to assess their efficacy and safety profile for the treatment of LV thrombus.

Limitations

This study is an observational, prospective analysis of a large cohort of patients, however the number of patients treated for LV thrombus and specifically with NOACs is small. However, this is the largest contemporary series assessing LV thrombus post STEMI and includes the largest number of patients treated with NOACs for this purpose.

The key limitations relate to the small sample size and the non-randomised nature of this study. This observational design means that despite efforts to correct for confounding variables, there are likely to be residual confounders that we have been unable to correct for. Additionally, be design we have used convenience sampling where it could be argued that there may have been risks of selection bias, however all attempts possible to avoid this have been made. In those patients with previous AMI it is possible the LV thrombus was long-

standing however in no cases was this felt to be the case at the time. Furthermore, we cannot extrapolate these findings to all NOACs because in out institution we did not use dabigatran for the treatment of LV thrombus and because of its different mechanism of action, we cannot extrapolate this data to its use for this indication. .

Conclusion

This hypothesis generating study suggests improved thrombus resolution in post ACS LV thrombosis in patients treated with NOACs compared to VKA. This improvement in thrombus resolution was accompanied with a better safety profile for the NOAC patients' versus VKA treated patients. This supports the need for randomised controlled trials to confirm this observational data.

Table 1: Clinical Characteristics in AMI patients comparing those with and without LV Thrombus			
	No LV Thrombus (n= 2,227)	**LV Thrombus** (n = 101)	**P value**
Age (mean+/-SD)	64.88±15.1	59.61 ±14.1	<0.001
Male, n (%)	1700 (76.3)	86 (85.1)	0.307
Past Medical History			
Current Smoker, n (%)	675 (30.3)	28 (27.7)	0.656
Hypercholesterolaemia, n (%)	802 (36.0)	38 (37.6)	0.823
Hypertension, n (%)	1033 (46.4)	45 (44.6)	0.796
Diabetes mellitus, n (%)	416 (18.7)	17 (16.8)	0.738
Prior Myocardial infarction, n (%)	376 (16.9)	16 (15.8)	0.894
Previous PCI, n (%)	285 (12.8)	11 (10.9)	0.682
Peripheral Vascular Disease, n (%)	68 (3.1)	2 (1.9)	0.749
Renal Failure, n (%)	192 (8.6)	9 (8.9)	0.936
LVEF (mean +/-SD)	46.1 ± 14.0	34.5 ±9.6	<0.001
Anterior infarction, n (%)	817 (36.7)	88 (87.8)	<0.001
Peak Troponin T (hs, ng/L) (median, IQR)	3100 (2400-7300)	7200 (2200-9000)	<0.001
Cardiogenic Shock, n (%)	180 (8.1)	8 (7.9)	0.898
Medical Treatment, n (%)	98 (4.4)	21 (20.7)	<0.001
Percutaneous Coronary Intervention, n (%)	2129 (95.6)	80 (79.3)	0.800
Drug-Eluting Stent Use, n (%)	2028 (95.2)	77 (96.2)	0.833

Table 2: Clinical Characteristics in Patients with Left Ventricular Thrombus who received anticoagulation (Warfarin/NOAC)			
	Warfarin (n= 60)	**NOAC** (n = 41)	**P value**
Age (mean+/-SD)	60.81±14.3	58.73 ±14.2	0.201
Male, n (%)	51 (85)	33 (80.4)	0.302
Past Medical History			
Current Smoker, n (%)	20 (33.3)	8 (21)	0.175
Hypercholesterolaemia, n (%)	19 (31.7)	19 (50)	0.150
Hypertension, n (%)	22 (36.4)	23 (60.5)	0.068
Diabetes mellitus, n (%)	10 (16.7)	7 (18.4)	0.999
Prior Myocardial infarction, n (%)	22 (36.7)	21 (55.3)	0.140
Peripheral Vascular Disease, n (%)	1 (1.7)	1 (2.6)	0.999
Renal Failure, n (%)	5 (8.3)	5 (12.1)	0.522
Mean Baseline eGFR (+/-SD_	66.11 ± 18.8	68.24 ± 15.8	0.230
Prior History of Malignancy	7 (11.6%)	6 (14.6%)	0.765
LVEF (mean +/-SD)	35.4 ± 9.0	33.5 ±10.0	0.854
Anterior infarction, n (%)	52 (86.7)	36 (87.8)	0.710
HAS-BLED Score (mean ± SD)	2.6 ± 1.0	2.7 ± 1.0	0.746
Medical Treatment, n (%)	12 (20.0)	9 (21.9)	0.850
Percutaneous Coronary Intervention, n (%)	48 (80.0)	32 (78.1)	0.800
Drug-Eluting Stent Use, n (%)	46 (95.8)	31 (96.7)	0.833
Medical therapy at D/C			0.688
Triple therapy	42 (70.0)	28 (68.3)	
Anticoagulation + Single antiplatelet	13 (21.7)	10 (24.4)	
Anticoagulation only	5 (8.3)	3 (7.3)	

Table 3: Clinical Characteristics in Patients with Left Ventricular Thrombus who received anticoagulation (Warfarin/NOAC)			
	Warfarin (n= 60)	**NOAC** (n = 41)	**P value**
Systemic thromboembolic Events			
Cerebral (CVA)	3 (5%)	1 (2.4)	0.388
Bleeding (BARC type, n (%)			
BARC 1	15 (25.0)	6 (14.6)	0.318
BARC 2	4 (6.7)	0 (0)	0.147
BARC 3a	1 (1.7)	0 (0)	1.000
BARC 3b	1 (1.7)	0 (0)	1.000
BARC 3c	1 (1.7)	0 (0)	1.000

Figure 1. Bar chart comparing rates of thrombus resolution between NOAC and Vitamin K antagonists. A significant unadjusted difference in rates of resolution was observed between patients on NOACs compared with patients who on vitamin K antagonists (p=0.002).

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