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Case Study

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DIRECT ORAL ANTICOAGULANTS FOR TREATMENT OF LEFT VENTRICULAR CLOT, A CASE SERIES AND LITERATURE REVIEW

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ABSTRACT

Five patients with the diagnosis of left ventricular (LV) clot, resulting from ischemic and non ischemic cardiomyopathy, were identified between February 2015 and February 2019, as they were deemed vitamin K antagonist's inappropriate because of the poor compliance with checking the level of anticoagulation. These patients were placed on direct oral anticoagulants (DOACs), and were followed clinically and by echocardiography for six months up to four years period. During such period, there has not been any identification of thrombo-embolic events to any of these patients, whereas echocardiography was performed at 1 and 3-6 months post initiation of DOACs.

INTRODUCTION

The diagnosis of LV clot is a common encounter in patients with regional or extensive LV dysfunction, especially post myocardial infarction.[1] It is noteworthy that 15-25% of the patients might develop LV clot post anterior myocardial infarction as a result of stasis, hypercoagulability and/or myocardial injury (Virchow triad).[2] The risk of thromboembolism may occur in 10-40% of such patients[3] if no anticoagulation was utilized, in which the current practice guidelines referenced vitamin K antagonist as the standard treatment to decrease the chance of thrombo-embolism.

The emergence of DOACs, such as dabigatran, rivaroxaban, edoxaban and apixaban, have proven to be superior to vitamin K antagonist in patients with non valvular atrial fibrillation (AF)[4]; however, there has not been any established guidelines for the use of such agents for LV clot.

CASE REPORT

Five patients were identified with LV clots between 2012 and 2018, in which four of them were two-six weeks post anterior wall myocardial infarction, whereas one of them was with dilated cardiomyopathy. All patients underwent transthoracic echocardiography as part of routine clinic visit. All five patients were treated with one of DOACs due to non-compliance with oral vitamin K antagonists. All patients were followed-up with clinic visits and echocardiography at 1, 3 and 6 months post diagnosis (see table 1).

Table 1.

Age/Sex	Basic Disease	Echocardiography Findings	DOACs (dose)	Other Medicine	Course	Thromboembolism
30/M	CAD HF	-LV apical clot (3.0x2.0 cm^2) -EF:45%	Apixaban (5 mg twice daily)	Aspirin	Stable	None
52/M	CAD HF	-LV apical clot (0.5x0.9 cm^2) -EF:42%	Apixaban (5 mg twice daily)	Aspirin	Resolved	None
41/M	CAD HF	-LV apical clot (2.5x2.0 $cm^{2)}$ -EF:35%	Apixaban (5 mg twice daily)	Aspirin	Resolved	None
67/M	CAD HF	-LV apical clot (1x1 $cm^{2)}$ -EF:42%	Rivaroxaban (10 mg once daily)	Aspirin	Resolved	None
50/M	Dilated Cardiomyopathy HF	-LV apical clot (1.5x1.0 $cm^{2)}$ -EF:20-25%	Apixaban (5 mg twice daily)	Aspirin	Improved (0.2x0.5 $cm^{2)}$	None

CAD: coronary artery disease, HF: heart failure

DISCUSSION

LV thrombus is not unusual in the setup of regional or diffuse LV systolic dysfunction, the combination of myocardial injury with subsequent wall motion abnormality in the setting of the hypercoagulable milieu, accompanying myocardial infarction, may lead to the development of LV clots (Virchow triad)(**Error! Bookmark not defined.**). Up to 40% of those patients might develop systemic thrombo-embolism 3, with noticeable increasing risk with advancing age, presence of atrial fibrillation, history of thrombo-embolism and poor compliance with anticoagulation.

Diagnosis of LV thrombus usually occurs as part of routine performance of trans-thoracic echocardiography, where sensitivity is 95%, specificity of 85-90% for detecting LV clots post myocardial infarction, which is defined by echocardiography as discrete echo dense mass with defined margins that are distinct from the endocardium, and seen in both systole and diastole, adjacent to hypokinetic or kinetic wall and detected in at least two views.[5]

Both current European society of cardiology and American College of Cardiology/American Heart Association (ACC/AHA) guidelines recommended vitamin K antagonist therapy for patients with LV clot post myocardial infarction. However, the slow onset of action, necessity for frequent monitoring, narrow therapeutic range, dietary restrictions, and frequent drug-drug interaction made vitamin K antagonist less attractive for many patients, in addition to the frustrations of both patients and doctors in achieving therapeutic anticoagulation levels when using such agents.[6]

On the other hand, DOACs do not have the aforementioned limitations, with less hemorrhagic risk; in fact, the 2014 American Heart Association stroke guidelines DOACs were given new class 2B recommendation level, evidence C for patients with LV mural clot intolerant to vitamin K antagonists to be used for 3 months.[7]

The available data in literature on the use of DOACs for LV clot came from sporadic case reports (**Error! Bookmark not defined.**,[8]) which showed resolution of LV thrombus; however, no data is yet available to show utility of these drugs in preventing LV clots in high risk patients.

In this cases series that we are reporting three out of five patients 60% had resolution of the clot with 80% improvement rate and 100% success rate in stabilizing and/or preventing thromboembolism (see table 1).

This case series shall shed more light on the utility of using DOACs in LV clot patients, who were deemed inappropriate to use vitamin K antagonists because of the poor compliance, and it shows their efficiency in causing regression or stabilization of LV clots and preventing thromboembolism over a long time of follow up, yet maintaining safety margins. This report is aimed at helping increase the amount of evidence which shall support the routine use of these agents for such patients.

CONCLUSION

DOACs may be safely used to treat patients with LV clots, especially if there are concerns regarding compliance or tolerance to vitamin K antagonists. More studies are required to advocate their routine use.

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