Executive summary of the SISET position paper on the management of antithrombotic therapy in left ventricular thrombus

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We here report a summary of a position paper from the Italian Society of Hemostasis and Thrombosis (SISET) on the antithrombotic management of patients with left ventricular thrombus (LVT).[1] The aim was to provide practical advice as the daily clinical management is hampered by the scanty available information.

Methodological features have been extensively reported in a previously published full paper.[1] Briefly, the wording "recommend" indicates a strong consensus among the experts (>90%) and/or the availability of high-quality evidence. The wording "suggest" reflects a weak guidance statement with moderate consensus among the experts (75% to 89%) and/or the availability of lower-quality evidence.

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Which are the epidemiology and risk factors for LVT?

Acute myocardial infarction represents the most relevant risk factor for LVT development with recent incidence values up to 4-15% of patients.[2,3] In this setting, the risk of LVT is greater in anterior wall or large area involvement, in cases of relevant delay to reperfusion-time or pre-angioplasty TIMI flow grade ≤1, and with a reduced left-ventricle ejection fraction. Other risk factors include atrial fibrillation, left-ventricle apical akinesia with aneurysm, left-heart valvular disease.[1]

LVT may also develop –with lower incidence– in patients with dilated cardiomyopathy, Takotsubo syndrome, left-ventricle non-compaction, peripartum cardiomyopathy, intracardiac devices or anti-cancer treatments, other forms of cardiomyopathy (e.g., hypertrophic cardiomyopathy, cardiac amyloidosis, Chagas disease, eosinophilic myocarditis).[1] The presence of high-risk features (e.g., left-ventricle dysfunction with or without segmental hypokinesia, presence of a scar and of a turbulent intracardiac flow) increases the risk of LVT development in these latter cases.[1]

Is anticoagulation effective in preventing LVT?

The available studies mostly regard patients with acute myocardial infarction and are variable in terms of patients' characteristics, antithrombotic therapy (i.e., low molecular weight heparin, vitamin K antagonists – VKAs; direct oral anticoagulants - DOACs), and time of publication. Overall, available evidence suggests a benefit in terms of LVT reduction in patients on anticoagulants that is however counterbalanced by a non-negligible risk of bleeding estimated to be as high as 4.7%.[1] Thus, the identification of patients in whom primary prophylaxis with anticoagulants may have a favorable risk-benefit profile is crucial but not well understood yet. The most recent available randomized trial found lower risk of LVT and doubled risk of bleedings in patients taking low-dose rivaroxaban (2.5 mg q12h) compared to

placebo.[4] However, methodological issues (e.g., inclusion of Chinese patients only with a preserved or moderately-reduced left-ventricle ejection fraction, high rate of drop-out) limit the generalizability and robustness of results.[4]

How to treat patients with LVT?

Anticoagulant treatment needs to be administered with the aim of LVT resolution and embolic stroke prevention. Evidence on the effectiveness and safety of heparins remains limited, while it is more robust for VKAs, and is progressively increasing for DOACs. Furthermore, most studies included patients with ischemic heart disease.[1] In case of VKAs administration, the suggested INR range would be 2.0-3.0 and the highest possible Time in Therapeutic Range (TTR) is advisable.[1] It should be acknowledged that a recent study reported lower incidence of ischemic events and higher rate of thrombus resolution even in patients with a TTR>50%.[5] The largest and up to date available meta-analysis reported a similar effectiveness and safety profile of DOACs compared to VKAs.[6] Most studies, however, had a retrospective design, a small sample size, and were heterogeneous in terms of outcome definitions and follow-up duration.[6] Waiting for additional data, the use of DOACs is primarily suggested in patients who are unsuitable for, or refuse, VKAs.[1]

How long should anticoagulant therapy be continued in patients with LVT?

LVT resolution –as well as embolic events– generally occur within the first 6 months of therapy. LVT recurrence may develop in 10-20% of patients after resolution.[1,7] The time (i.e., recent), the type (i.e., protuberant), and the size (i.e., smaller) of LVT appeared to be associated with an earlier resolution. Conversely, a reduced left-ventricle ejection fraction and a left-ventricle apical aneurysm appeared to be associated with a lower odd of resolution.[8] Longer treatments with anticoagulants may be useful in higher-risk patients, such as those with reduced left-ventricle ejection fraction or left-ventricle aneurysm. Recent data, indeed, reported higher incidence of LVT resolution in patients receiving 12 than 3 and 6 months of anticoagulation.[8]

May anticoagulant therapy safely be administered along with antiplatelets?

Dual antithrombotic therapy may be needed in specific subgroups of patients (e.g., those needing reperfusion therapy), but it is associated with an increased bleeding risk. In these cases, the therapeutic management may be extrapolated from available ran-

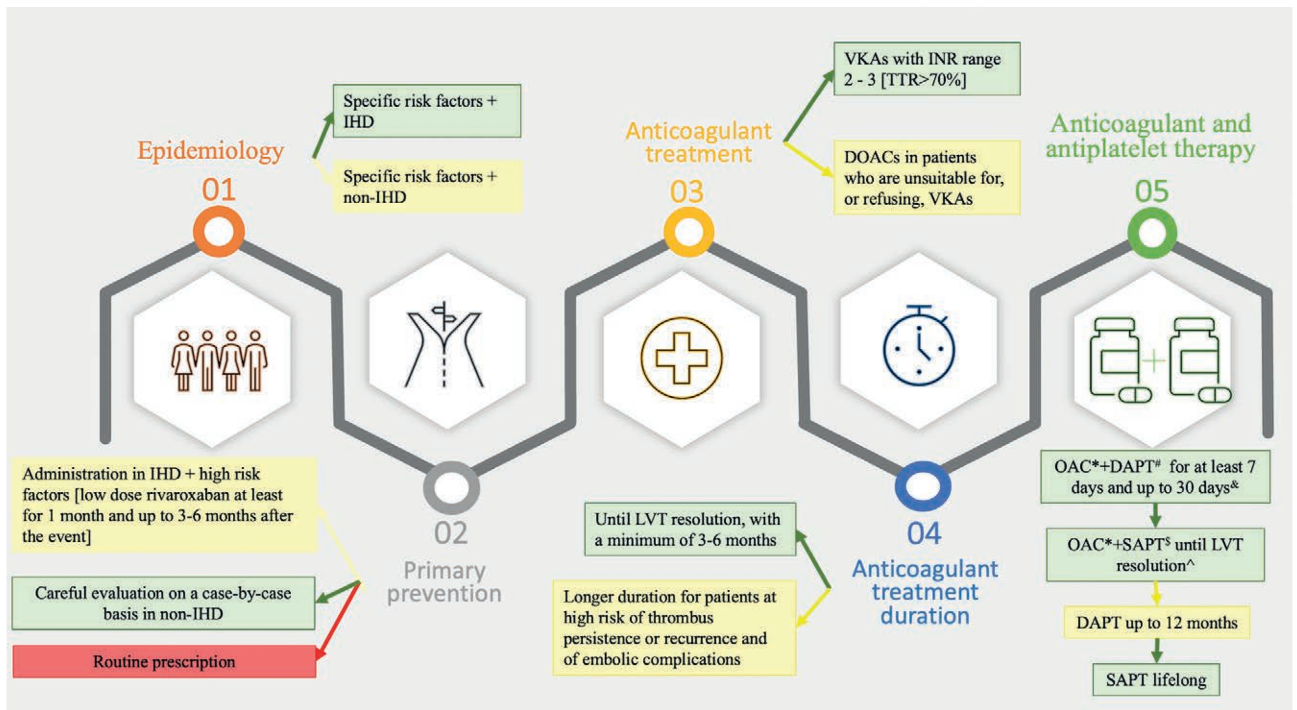

Figure 1. Executive summary of the position paper's statements. The figure summarizes the statements provided for the five questions addressed in the position paper. Green arrows indicate a recommendation ("is recommended"), yellow arrows indicate a suggestion ("is suggested"), and red arrows denote a recommendation against ("is not recommended"). *VKAs, evaluate DOACs in selected cases (see text); #clopidogrel is preferred as P2Y12 inhibitor; $P2Y12 inhibitor is preferred; ^evaluate longer OAC administration if thrombus persistence and/or high-risk features; &it may be useful to consider OAC + SAPT in patients with a high risk of bleeding and in medically-managed acute coronary syndrome. DAPT, dual antiplatelet treatment; DOACs, direct oral anticoagulants; IHD, ischemic heart disease; INR, international normalized ratio; LVT, left ventricular thrombus; OAC, oral anticoagulant therapy; SAPT, single antiplatelet therapy; TTR, time in therapeutic range; VKAs, vitamin k antagonists.

domized trials on LVT.[9,11] Briefly, triple therapy with an oral anticoagulant and two antiplatelet drugs is administered for 7-30 days and is followed by dual therapy with an oral anticoagulant and a single antiplatelet drug (P2Y12 inhibitor is preferred). The oral anticoagulant is replaced by aspirin at LVT resolution (or when the anticoagulant is withheld) and dual antiplatelet treatment is continued. At 12 months from the index event, treatment with a P2Y12 inhibitor only is continued indefinitely.[1] In patients with a very high risk of bleeding –and in those with medically managed acute coronary syndrome– it may be appropriate to consider using anticoagulation combined with a single antiplatelet agent rather than triple-therapy regimens.[1]

Conclusions

Due to the absence of a clearly established, evidence-based approach for identifying and treating patients with LVT, a comprehensive assessment of individual risk factors remains essential. Although the best management strategy is still under investigation, this position paper offers practical guidance for the management of LVT in routine clinical settings (Figure 1).

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