Thrombotic complications

Prof James Ker, Senior Lecturer, Department of Internal Medicine, University of Pretoria

There have been significant advances in the treatment of heart failure (HF) with a reduction in mortality. Despite this improvement in mortality, HF still has a bad prognosis and research is ongoing to find ways to improve the prognosis.

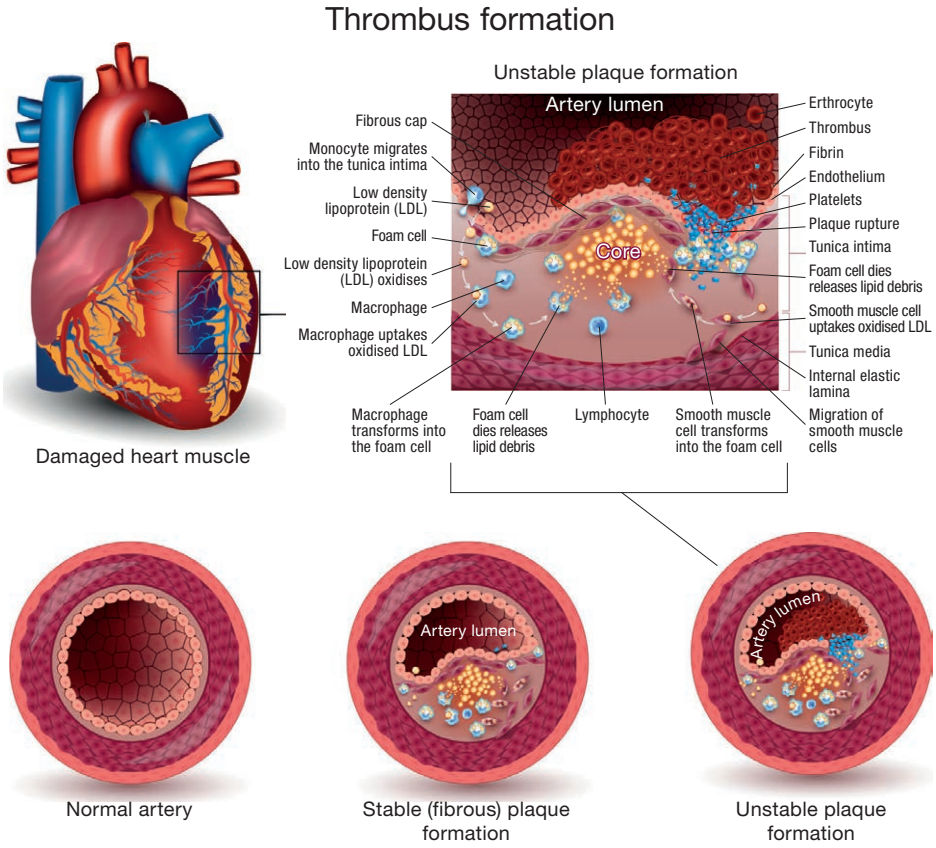
Accumulating evidence indicate that thrombotic complications may play a major role in morbidity and outcomes in patients with HF. There are two major sources of thrombosis in HF.

The one important source is atrial fibrillation (AF) that is common in HF and the role of oral anticoagulation is well established in AF, but the role of anticoagulation is less well established in HF without AF. The other source of thrombosis in HF is venous thromboembolism (VTE).

Hospitalised HF patients have an increased risk of VTE as a result of vascular abnormalities, increased coagulability and impaired blood flow. Endothelial damage and dysfunction is well documented in HF and is considered a hallmark of HF with severity increasing with severity of HF.

Venous dysfunction has also been described in HF and together with stasis of venous blood in oedematous legs sets the stage for thrombosis. The incidence of VTE in these patients is reported to be as high as 26% especially in those patients with decompensated HF.

It has also been shown that HF increases the risk of pulmonary embolism (PE) that is not associated with deep vein thrombosis. The contribution of VTE towards overall mortality in HF is not minor. It has been shown in a meta-analysis that one in five deaths occur in HF patients on angiotensin-converting-enzyme inhibitors. Indeed, post-mortem studies show that PE can occur in about 30% of patients with HF. However the relationship between HF severity and VTE is not well established.



NEW STUDY

In this study, the MAGELLAN study, with 2593 HF patients, New York Stage 111 or 1V, were evaluated for VTE. The results of this study showed that the more severe HF patients had a greater incidence of VTE up to day 10 and day 30 in hospital. This study also showed that this link between the severity of HF and the risk of VTE may be disrupted with oral anticoagulants (rivaroxaban).

Should we consider the use of oral anticoagulants in patients with severe HF?

REFERENCES

Haas S. Venous thromboembolism in medical patients: the scope of the problem. *Semin Thromb Hemost* 2003; 29 (Suppl. 1: 17-21.

Shantsila E, Lip GYH. Thrombotic complications in Heart Failure: an underappreciated challenge. *Circulation* 2014; 130: 387-389.

Sorensen HT *et al.* Heart disease may be a risk factor for pulmonary embolism without peripheral deep venous thrombosis. *Circulation* 2011; 124: 1435-1441.

Garg R *et al.* Overview of randomized trials of ACE-inhibitors on mortality and morbidity in patients with heart failure. *JAMA* 1995; 273: 1450-1456. SF