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Letter on ‘Idarucizumab for the reversal of dabigatran in patients undergoing heart transplantation’

Van Keer *et al.*¹ in their work ‘Idarucizumab for the reversal of dabigatran in patients undergoing heart transplantation’ presented a new appealing approach for managing patients on anticoagulants and waiting for heart transplantation (HTx). Atrial fibrillation (AF) is the main reason for anticoagulants, as this is the most common arrhythmia in chronic heart failure (HF), which increases proportionally with the severity of HF. The biggest dilemma in these patients is to provide the optimal prevention therapy with a balanced safety profile, and the ability to quickly and totally reverse anticoagulation when needed, particularly if a tremendous surgical procedure is waited in the near but unknown future. Dabigatran, with development of idarucizumab, has become a more convenient anticoagulation option as it can be effectively reversed prior to HTx. In our institution we had a similar experience in the past year, with a cohort of eight patients treated with dabigatran (seven for AF and one for venous thrombosis) who underwent HTx after having received 5 g idarucizumab. Similarly, in our cohort, the patients had various aetiologies of HF, with a median age of 58.7 ± 7.0 years, a median CHA₂DS₂-VASc score of 2.0 and a mean estimated glomerular filtration rate of 47.0 ± 24.2 mL/min. All patients were

fully anticoagulated prior to surgery and the routine coagulation panel with pro-thrombin time, activated partial thromboplastin time and thrombin time was indicative for the *in vivo* effect of dabigatran. The HTx procedures were performed without excess of bleeding, with a low use of blood products and coagulation factors (mean autologous transfusion 0.62 ± 0.29 L, mean of red blood cells during and post-procedures 0.63 ± 0.86 and 0.38 ± 0.70 , respectively). The median blood products used during hospital stay (red blood cells, fresh frozen plasma and clotting factors) were 2.50 ± 2.69 units. No graft failure was observed, none of the patients required re-thoracotomy due to bleeding complications, no thrombotic complications occurred, and all patients were alive and well after 90 days of follow-up. One would argue why to pursue a risky strategy when these patients are already complicated and heart grafts are such a scarce resource. On the other hand, there is evidence in the literature that dabigatran and vitamin K antagonists are associated with similar rates of periprocedural bleeding, including patients having urgent surgery, but hemostasis control is achieved better pre-operatively with dabigatran–idarucizumab.² Furthermore, the availability of idarucizumab in settings of emergency surgery/urgent procedures or life-threatening or uncontrolled bleeding is proven safe.³ By using the dabigatran–idarucizumab strategy, we offer our patients the best optimal preventive treatment allowing them to survive to HTx. The main limitation of these two cohorts is the small number of patients over long time periods, with some confounding factors that limit the analysis of this strategy. The main bias in this paper remains the two

different periods for considering patients receiving vitamin K antagonists or dabigatran. In conclusion, we agree with our colleagues that this strategy should be a choice for non-valvular AF patients awaiting HTx, but more clinical trials are needed to confirm these data and better define clinical guidelines.

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