Levosimendan in acute and advanced heart failure: still some chapters to be written

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The essays in this Supplement to the European Heart Journal emerged from a series of tutorials delivered at the 2018 congress of the European Society of Cardiology (ESC), convened in Munich, Germany. It must be acknowledged at the outset that scope and range of issues addressed during these tutorials considerably exceeded the contents of this Supplement. This is a reflection of the fact that the management of acute heart failure (AHF) and advanced heart failure (AdHF) are among the most complex and challenging aspects of caring for patients with heart failure.

Our focus on current expert thinking and best practice in the use of levosimendan in AHF and AdHF is aligned to important wider trends in thinking about the management of these conditions, some of which found recent expression in the work of Dr Maria Crespo-Leiro et al. of the Heart Failure Association of the ESC. The first of their salient conclusions is AHF and AdHF are both, at their core, states of decompensation and instability.

As a first-in-class inodilator that enhances cardiac contractility though calcium sensitization and exerts vasodilator and cardioprotective effects via opening of ATP-dependent potassium channels in vascular smooth muscle cells and mitochondria, levosimendan is increasingly well regarded as one of the options that may be drawn on in these unstable situations when standard therapies fail to control symptoms and when systemic haemodynamics and organ perfusion and function may be jeopardized.

Crespo-Leiro et al.—and several contributors to this Supplement—identify other aspects of levosimendan that are relevant to AHF and AdHF. Firstly, levosimendan differentiates profoundly from the catecholaminergic inotropes not only in its pharmacologic efficacy but also in its safety parameters: there is no indication that it shortens life. This contributes to the increasingly widespread view that a general ‘de-catecholaminization’ of unstable and critically ill patients may be a goal in its own right.

Second, levosimendan exhibits a long duration of action (~7 days), courtesy of an active metabolite. This quality is directly pertinent to the use of intermittent use of levosimendan for long-term stabilization of symptoms, a practice that has moved towards the clinical mainstream in recent years, propelled by experience in studies such as LEVOR-Rep, LION-HEART, and LAICA, all of which are examined in this Supplement.

The central consideration here, and one that I wish to highlight in this Editorial, is that patients of the kind we are concerned with are vulnerable to repeated cycles of decompensation and hospitalization. Such episodes are debilitating for the patient and expensive for the health system. If we can intervene to restore equilibrium to patients in the vital time between the start of a phase of deterioration and the eventual hospitalization we could contribute significantly to their health-related quality of life and spare them the disappointment of yet another unscheduled hospital admission. In conditions as complex and variable as AHF or AdHF it would be foolish to claim that any one intervention can be the silver bullet that cures all ills; but, as I believe the contents of this Supplement demonstrate, there is certainly enough evidence to identify levosimendan as one valuable element in an overall strategy of care directed towards maintaining patients in a condition of out-of-hospital stability.

Reference


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