Letters to the Editor

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Lessons from EuroHeart Failure Survey

In some instances, acute myocardial infarction (AMI) may be the precipitating factor both for acute heart failure (AHF) and for diabetic ketoacidosis (DKA), with the consequence that the two disorders may occasionally co-exist in the same individual.1,2 When de novo AHF has cardiogenic pulmonary oedema as its presenting feature, its co-existence with DKA can pose unique diagnostic and therapeutic challenges. On the one hand, if the onset of AMI has escaped detection, due to a pain-free presentation, it may be difficult to differentiate between AMI-related cardiogenic pulmonary oedema and DKA-related adult respiratory distress syndrome, a diagnostic dilemma compounded by the fact that stigmata such as ST-segment elevation and a rise in cardiac troponin levels may be a feature, not only of AMI, but also of DKA per se.3 In the context of undisputable de novo AHF, the therapeutic challenge is one of managing cardiogenic pulmonary oedema and its aftermath, chronic heart failure, with minimal use of diuretics so as to reduce the risk of activation of the renin–angiotensin–aldosterone system (RAAS),4 with its attendant adverse sequelae.5 Although participants in the EuroHeart Failure study managed cardiogenic pulmonary oedema with intravenous diuretics and with intravenous nitrates, in 94 and 70.6% of instances, respectively,6 implying co-prescription of the two agents in some of those instances, the ideal strategy may well have been the sole use of intravenous nitrates7 given the fact that patients with new-onset cardiogenic pulmonary oedema are unlikely to have a net increase in blood volume, the latter eventuality rendered even less likely by the co-existence of DKA. Following the resolution of pulmonary oedema, the subsequent management of these patients should, therefore, prioritize the use of angiotensin-converting enzyme inhibitors, as opposed to diuretics, so as to continue ‘unloading’ the left ventricle without activating the RAAS. Unless contraindicated, these agents should be co-prescribed with aldosterone antagonists8 to a greater extent than was the case in the Euro Heart Failure study.6 Through the mediation of aldosterone blockade, such a strategy would mitigate the adverse sequelae of RAAS activation. Furthermore, by blocking tubular re-absorption of sodium at multiple sites, co-prescription of the two agents could, in theory, also enhance natriuresis,9 thereby making it easier to use diuretics sparingly, should they subsequently prove to be necessary. In the latter eventuality, torasemide might well be the loop diuretic of choice, given the fact that it also possesses anti-aldosterone properties.10 In conclusion, in the context of de novo AHF, especially when it co-exists with DKA, there are huge opportunities to explore the paradigm of minimal use of diuretics in the acute, as well as in the chronic, phase of treatment.

References


Right ventricular involvement in Takotsubo cardiomyopathy

We read with great interest the study by Hagi et al.1, which confirmed that right ventricular (RV) involvement is common in Takotsubo cardiomyopathy (TTC) and seems to be associated with a more severe impairment in left ventricular (LV) systolic function. It may be suspected by the presence of pleural effusion. The methods and interpretation of the results, however, raise several concerns.

In this study, Hagi et al.1 report that nine patients had RV wall motion abnormality (WMA) on CMR imaging, in whom relevant history included hypertension, hypercholesterolaemia, diabetes, chronic obstructive pulmonary disease, osteoporosis, goiter, Graves’ disease, and paroxysmal atrial fibrillation. It is well known, however, that hypertension, hypercholesterolaemia, diabetes, chronic obstructive pulmonary disease, Graves’ disease, and paroxysmal atrial fibrillation could affect LV or RV myocardial segments and global function to some extent. How could the authors discriminate RV WMA of the nine patients caused either by TTC involving RV or by the above-mentioned diseases? If the explanation is that eight of nine patients with RV involvement had a follow-up study demonstrating complete recovery or significant improvement of the initial regional WMA, is there a relation of complete recovery or significant improvement of the initial regional WMA to the above-mentioned diseases to be optimal cured which could not be well described?

An echocardiography study of Lopez-Candales et al.2 certify that maximal tricuspid annular plane systolic excursion which could well reflect RV function is not only determined by RV systolic function but also appears to depend on LV systolic function. That is to say, RV WMA on CMR imaging could be affected by LV systolic dysfunction.