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. 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. 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), with its attendant adverse sequelae. Although participants in the Euro-Heart Failure study managed cardiogenic pulmonary oedema with intravenous diuretics and with intravenous nitrates, it is unlikely to have been the sole use of intravenous nitrates 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 antagonists to a greater extent than was the case in the Euro Heart Failure study. 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, 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.

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


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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 in this study, 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 López-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.

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Haghi et al. also think that RV involvement in TTC seems to be associated with a more severe impairment in LV systolic function, which could affect the precise evaluation of prevalence of RV involvement in TTC and further the prehension of pathophysiological mechanisms of TTC. RV WMA condition in patients with TTC and normal LV function needs to be studied.

References

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Right ventricular involvement in Takotsubo cardiomyopathy: reply

We thank Ze-Zhou Song and Jing Ma for their interest in our work. We are unaware of any data showing that hypertension, hypercholesterolaemia, diabetes, Graves’ disease, or paroxysmal atrial fibrillation can cause regional right ventricular wall motion abnormalities, although some of these conditions may affect global parameters of ventricular function. However, pulmonary hypertension which can be encountered in chronic obstructive pulmonary disease may cause regional right ventricular dysfunction. In fact, we have observed reversible akinesis of the right ventricular apex indistinguishable from right ventricular dysfunction of Takotsubo cardiomyopathy in a case of pulmonary embolism. As exacerbation of obstructive pulmonary disease was the triggering event in two of our patients, we cannot exclude that acute pulmonary hypertension has contributed to the observed right ventricular wall motion abnormalities in those two patients.

We did not specifically look for changes of treatment regimen upon follow-up cardiovascular magnetic resonance imaging. These data were only available for those three patients who had their follow-up examination within 10 days of admission. In these three patients, there was no change of treatment with regard to their co-morbidities.

The idea that left ventricular dysfunction in itself may cause right ventricular dysfunction is intriguing. In fact, this issue is currently under investigation at our institutions and results will shortly be available.

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New risk factors of heart failure?

We appreciate very much Siirilä-Waris et al. for their excellent work on risk factors of heart failure. Progression of heart failure may be due to an initial cardiac injury, or mutation of the genetic programme, in association with activation of neurohormones and proinflammatory cytokines, resulting into immune activation, which worsens heart failure. Therefore, it may be proposed that any factor which can block neuroendocrine activation would be protective, whereas other factors that can enhance neurohormonal activity would be the risk factors of heart failure. Decreased heart rate variability, increased blood pressure variability, dyslipidemia, increased IL-6, IL-1, TNF-alpha, C-reactive protein, and adhesion molecules are other important determinants of mortality in patients of heart failure. Presence of coronary artery disease is also an important risk factor of mortality, which becomes worst if there is coexisting cardiac cachexia or obesity among these patients.

Apart from above risk factors, nutritional factors such as increased consumption of proinflammatory foods; refined starches and sugar, trans fatty acids, w-6 fatty acids, and saturated fat may enhance proinflammatory cytokines. Therefore eating proinflammatory foods could be an important cause of increased mortality in heart failure, because these patients have a pre-existing proinflammatory milieu. These foods may produce oxidative stress, free fatty acids, and proinflammatory substances, which result in endothelial dysfunction. Glucose ingestion in normal subjects is associated with increased superoxide generation in leukocytes and mononuclear cells, as well as with raised amount and activity of nuclear factor-κB (NF-κB), a transcriptional factor regulating the activity of at least 125 genes, most of which are pro-inflammatory. Increased consumption of refined carbohydrates also causes an increase in two other proinflammatory transcription factor, activating protein–1 (AP-1), and Erg-1, the first regulating the transcription of matrix metallo-proteinases and the second modulating the transcription of tissue factor and plasminogen activator inhibitor-1. These adverse factors related to diet may worsen the prognosis in heart failure.

A mixed meal from a fast-food chain has also been shown to induce activation of NF-κB associated with the generation of reactive oxygen species (ROS) by mononuclear cells. Superoxide anion appears to be an activator of at least two major proinflammatory transcription factor, NF-κB and AP-1. These observations are consistent with previous findings, demonstrating that after oral or intravenous glucose challenges, in both normal subjects and patients with type 2 diabetes mellitus, there is an increased generation of ROS and raised circulating levels of proinflammatory cytokines, such as TNF-α, IL-6, and IL-18. In apparently healthy subjects, a single high-fat meal produces endothelial activation, as evidenced by increased concentrations of the adhesion molecules VCAM-1 (vascular cell adhesion molecule-1) and ICAM-1 (intercellular adhesion molecule-1), in association with raised plasma concentrations of IL-6 and TNF-α. A high-fat meal may increase the circulating levels of...