Letters to the Editor

Reply to Letter to the Editor "Troponin I, cardiac ventricular dysfunction and causal toxicity of chemotherapy drugs", by L. Delval (Ann Oncol 2002; 13: 1952–1953)

We thank Dr Delval for her interest in our work. We recognise that left ventricular (LV) dysfunction can occur in critical patients with septic shock. Although localised and systemic infection was frequently observed in our study population after high-dose chemotherapy (HDC), no patient developed septic shock or prolonged hypovolaemia-related hypotension requiring intensive care unit admission. Furthermore, all patients underwent echocardiographic evaluation before every cycle of HDC (i.e. every 28 days during the treatment period) and in all cases cardiac function was within the normal range; indeed, a normal cardiac function is required to continue HDC. Therefore, in the absence of early LV dysfunction it is very unlikely that a sepsis-associated cardiac problem occurred. The incidence of noncritical episodes of sepsis during the hospitalisation period was equally distributed between troponin I (TnI)-positive and TnI-negative patients. We did not observe episodes of either sepsis or myocarditis during the long-term follow-up.

We agree that TnI "is not a specific marker for the origin of the cardiac dysfunction", neither does it give us information about the aetiopathogenesis of its release, but this was not the aim of our study. The exact mechanism of TnI release after HDC remains to be elucidated and, besides direct drug toxicity effects, other contributing causes cannot be excluded. Nevertheless, the clinical relevance of TnI release remains unchanged. In fact, it allows the identification of high-risk cardiac patients after HDC and the prediction of the degree of LV ejection fraction reduction. The message of our study is simple and practical: detection of a TnI-positive value, regardless of the mechanism involved, is an important warning.

We well know that the primary aim is to treat cancer. However, this purpose should be reached without unacceptable cardiac side effects. In this setting, TnI-related information should not be utilised to reduce the oncological therapeutical opportunities of patients; rather, TnI should be considered a simple and low-cost marker of cardiac injury that allows stratification of cardiac risk after HDC. On this basis, close cardiac monitoring, cardiological support, and the use of cardioprotective agents are possible future approaches in need of further investigation.

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DOI: 10.1093/annonc/mdf344