Cardiotoxicity: Risk Assessment and Prevention

Hyukjin Park
Chonnam National University Hwasun Hospital / Medical School, Korea

The therapeutic landscape for haematologic malignancies has undergone a paradigm shift. While traditional agents like anthracyclines remain an important treatment option, the emergence of proteasome inhibitors, BTK inhibitors, BCR-ABL TKIs, and various immunotherapies has significantly extended patient survival. However, this success has brought a parallel increase in the prevalence and morbidity of cancer therapeutics-related cardiovascular toxicities (CTR-CVT). In the era of 'survivorship', the challenge for haematologists is no longer just treating the cancer, but managing the long-term cardiovascular (CV) consequences.

Despite being decades old, anthracycline-induced CVT remains a significant clinical burden. As patients survive their initial malignancy and move on to subsequent lines of therapy, the cumulative cardiac damage often manifests years later. Moreover, it is well known that 'novel' treatment options described above also carries substantial risk of various CV diseases as well. This increasing pool of survivors at risk necessitates a more vigilant, long-term approach to cardiac monitoring than previously practiced.

The 2022 ESC guidelines on Cardio-oncology introduced the HFA-ICOS risk assessment tools to provide a standardized framework. While these tools are fundamental, they possess inherent limitations: they are increasingly complex to apply in busy clinical settings, do not cover all emerging haematologic agents, and may not precisely predict risk for every patient. Nevertheless, the goal of these tools is clear: early detection. Wating for symptomatic heart failure or dramatic drop in LVEF often leads to irreversible damage, permanent cessation of life-saving haematologic treatment, and increased mortality.

Currently, there is no universal magic bullet for the primary prevention of CTR-CVT. While the STOP-CA trial demonstrated the benefits of atorvastatin in preventing anthracycline-induced CTRCD in patients with lymphoma, the most effective strategy remains the aggressive management of underlying traditional CV risk factors. Optimising hypertension, diabetes, and dyslipidaemia is not simply about improving a percentage of ejection fraction, rather, it is about widening the therapeutic window, allowing patients to tolerate and complete their planned oncologic regimens without interruption. A diagnosis of CVT should not automatically signal the end of haematologic therapy. With modern heart failure medications and close collaborative monitoring, many patients can safely treat their malignancy. And even after recovery from CTR-CVT, they should remain on cardioprotective therapy without drug withdrawal.

The ultimate goal of cardio-oncology in haematology is to ensure that CVTs never become the limiting factor in a patient's cancer therapy. By focusing on early risk stratification, aggressive prevention of traditional risks, and committed long-term cardiac management, we can improve both the oncologic outcomes and the quality of life for our patients.