With the emergence of novel molecularly targeted agents and immunotherapeutics, cardiac and cardiovascular toxicities may occur as a consequence of on-target or off-target effects, including ventricular dysfunction, hypertension and QTc prolongation. Drug-induced ventricular dysfunction associated with anticancer agents occurs via distinct mechanisms. Cytotoxic chemotherapy agents such as anthracyclines can cause type I injury, which is characterized by myocyte cell death and is typically cumulative dose-related and permanent. In contrast, cardiac toxicity induced by molecularly targeted agents such as trastuzumab is classified as type II injury, and is due to myocyte dysfunction without cell death. As such, type II cardiac injuries are generally not cumulative dose-related and are predominantly reversible. Amongst the newer molecularly targeted agents, ventricular dysfunction has been reported with pertuzumab, trastuzumab-DM1, angiogenesis inhibitors including multikinase inhibitors and anti-DLL4 antibodies, ABL inhibitors, and MEK inhibitors. Immune-mediated and autoimmune myocarditis has been reported with immune checkpoint inhibitors such as anti-CTLA4 antibodies. Hypertension has been commonly reported with angiogenesis inhibitors and is also induced by MEK inhibitors in some patients. Prolongation of the QTc interval has been reported with HDAC inhibitors, ABL inhibitors, MET inhibitors and angiogenesis inhibitors. The pathophysiology, diagnosis and management of these cardiac and cardiovascular toxicities of novel anticancer agents will be discussed.

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