The prognostic power of two-dimensional strain-derived left ventricular myocardial work for all-cause mortality in pancreatic ductal adenocarcinoma patients receiving cancer therapy

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Background: Left ventricular (LV) myocardial work (MW) is a relatively novel parameter derived from the integration of LV global longitudinal strain (GLS) and LV pressure. MW may provide additive information beyond LV ejection fraction (EF) and GLS. However, the clinical implication of MW in pancreatic ductal adenocarcinoma (PDAC) patients receiving cancer therapy is still unclear.

Purpose: We conducted this prospective cohort study to investigate the clinical implication and the prognostic value of LV MW in PDAC patients receiving cancer therapy.

Methods: We prospectively enrolled a total of 234 newly diagnosed PDAC patients (65.1 ± 11.1 years old, 51% male). We did echocardiographic study at the following timepoints: before chemotherapy and 3 months after cancer therapy. MW parameters, including global work index (GWI), global constructive work (GCW), global waste work (GWW) and global work efficiency (GWE), would be determined from the pressure-strain loop. A ≥15% reduction in absolute GLS value from baseline was defined as signifying cancer therapy–related cardiac dysfunction (CTRCD). The primary outcome was defined as all-cause mortality.

Results: A total of 185 patients (64.7 ± 11.3 years old, 49% male) were included for analysis. We excluded 49 cancer patients who did not have echocardiographic studies at 3 months after cancer therapy. Over a median follow-up of 8 months, twenty-six (14.1%) patients had CTRCD and sixty (32.4%) patients were dead. There was no significant difference of baseline demographic and echocardiographic parameters between the survivors and the mortality patients. At 3 months after cancer therapy, compared to the survivors, the mortality patients had lower LV EF (survivor vs. death: 68.6 ± 6.5% vs. 63.5 ± 11.3%, p = 0.002) and lower GLS (survivor vs. death: -18.8 ± 2.2% vs. -17.3 ± 3.4%, p = 0.003). Moreover, the mortality group had lower GWI (survivor vs. death: 1984.2 ± 376.0 mmHg% vs. 1746.8 ± 495.1 mmHg%, p = 0.002) and GCW (survivor vs. death: 2307.0 ± 405.6 mmHg% vs. 2125.1 ± 493.9 mmHg%, p = 0.02). After adjusting for age, gender, LVEF (at 3-month), GLS and CTRCD, GWI < 1700 mmHg% was independently associated with all-cause mortality (hazard ratio 2.35, 95% confidence interval 1.37–4.03, p < 0.001; Figure).

Conclusion: Approximately 14% of PDCA patients receiving cancer therapy had CTRCD. Lower values of LV GWI (< 1700 mmHg%) was independently associated with increased all-cause mortality at a median follow-up of 8 months. LV GWI showed the better performance for mortality prognostic power compared to LVEF and LV GLS in PDCA patients receiving cancer therapy.
High GWI (>1700)

Low GWI (≤ 1700)

$p < 0.001$