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Original Article

Analysis of the effectiveness of the myocardial work index in the assessment of cancer therapy-related cardiac dysfunction

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Abstract: Objective: To investigate the correlation between myocardial global work index (GWI) and cancer therapy-related cardiac dysfunction (CTRCD) during chemotherapy for patients with thoracic tumors. Methods: A retrospective analysis was conducted on 87 patients with thoracic tumors who underwent chemotherapy at The Third Hospital of Hebei Medical University from April 2022 to June 2023. Among them, 37 patients who developed CTRCD during chemotherapy were assigned to the research group, and the remaining 50 patients without CTRCD were assigned to the control group. Myocardial GWI was compared between the two groups at three time points: before chemotherapy (T0), during chemotherapy cycle 3 (T1), and upon completion of chemotherapy (T2). The predictive value of myocardial GWI at T0 and T1 for CTRCD was analyzed. Patients were followed up for prognosis, and the relationship between myocardial GWI at T2 and the recovery from CTRCD was evaluated. Additionally, the correlation between myocardial GWI and Creatine Kinase-MB (CK-MB) and cardiac troponin T (cTnT) at T0-T2 was analyzed. Results: The myocardial GWI in the research group was lower than that in the control group at T0-T2 ($P < 0.05$). Myocardial GWI at T0 and T1 showed excellent predictive accuracy for the occurrence of CTRCD. In addition, patients in the research group who recovered from CTRCD had a higher myocardial GWI at T2 compared to those who did not ($P < 0.05$). The myocardial GWI was negatively correlated with CK-MB and cTnT at T0-T2 ($P < 0.05$). Conclusion: Myocardial GWI is a reliable predictor for the occurrence of CTRCD during chemotherapy in patients with thoracic tumors, providing valuable clinical insights.

Keywords: Myocardial global work index, cancer therapy-related cardiac dysfunction, creatine kinase-MB, cardiac troponin T, thoracic tumors, chemotherapy

Introduction

Tumors remain one of the leading causes of morbidity and mortality worldwide. Statistics show that over 20 million new tumor cases are diagnosed annually, with approximately 2.57 million deaths attributed to cancer each year [1]. Among these, thoracic tumors account for more than 60% of all cancers, including lung cancer, breast cancer, and esophageal cancer [2, 3]. Currently, surgery combined with radiotherapy remains the primary treatment for tumors in clinical practice. Since thoracic tumors are closely related to the heart, both radical resection and the toxic effects of chemotherapy drugs can adversely affect cardiac function [4]. It is reported that over 70% of patients with thoracic tumors experience varying degrees of cancer therapy-related cardiac dysfunction (CTRCD), significantly increasing the risk of poor prognosis [5]. Therefore, it is crucial to diagnose CTRCD early and accurately during chemotherapy in patients with thoracic tumors and implement timely, targeted interventions to promote recovery.

Echocardiography is the most commonly used clinical tool to assess cardiac function, with parameters such as left ventricular ejection fraction (LVEF) and global longitudinal strain (GLS) frequently used to evaluate CTRCD in patients [6]. However, an increasing number of

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studies have pointed out that LVEF and GLS are partially dependent on cardiac load. In some senior patients with impaired myocardial contraction, myocardial contractility may decrease, even though LVEF and GLS remain within the reference range [7, 8]. This can lead to an increased risk of missed or incorrect diagnosis. The myocardial global work index (GWI) refers to the area of the left ventricular pressure-strain loop (LV-PSL) and reflects the work done by the myocardium from mitral valve closure to mitral valve opening, indicating the metabolism and oxygen consumption of the left ventricle. As an afterload-corrected parameter, myocardial GWI exhibits superior independence compared to LVEF and GLS. Studies have shown that myocardial GWI changes more rapidly in response to left ventricular remodeling caused by coronary heart disease and structural heart disease [9, 10]. These findings suggest that myocardial GWI may offer superior efficacy in early diagnosis of CTRCD. However, there are currently no clinical reports to confirm this hypothesis.

If the relationship between GWI and CTRCD can be demonstrated, it could enhance the safety of chemotherapy for future tumor patients. Therefore, the present study aims to perform a preliminary analysis of the correlation between GWI and CTRCD during chemotherapy for thoracic tumors. This study pioneers the investigation to validate the relationship between GWI and CTRCD in patients undergoing chemotherapy for tumors, and its results may provide new insights and guidelines for assessing chemotherapy safety in the future.

Materials and methods

Patient selection

A total of 87 patients with thoracic tumors who underwent chemotherapy at The Third Hospital of Hebei Medical University between April 2022 and June 2023 were selected for retrospective analysis. Of these, 37 patients who developed CTRCD during chemotherapy were assigned to the research group, while the remaining 50 patients without CTRCD were assigned to the control group. This study was conducted in strict accordance with the *Declaration of Helsinki* and approved by the Ethics Committee of Third Hospital of Hebei Medical University. The study flow is shown in **Figure 1**.

Inclusion and exclusion criteria

Inclusion criteria: Age between 18 and 70 years; normal left ventricular systolic function (baseline LVEF ≥ 55%); no typical signs of cardiac failure before the start of anticancer therapy. Exclusion criteria: Expected survival < 6 months; active or prior cardiac diseases (myocardial infarction, unstable angina, persistent atrial fibrillation, moderate or severe regurgitation, or stenotic heart valve disease); autoimmune disorders, or vascular diseases.

Determination of results

Sinus tachycardia [11]: Defined as a heart rate exceeding 100 beats per minute in adults. Electrocardiogram (EKG) revealed a P wave frequency greater than 100 beats per minute, with P waves often overlapping with T waves, followed by the QRS wave after the P wave. Patients typically experience palpitations and precordial discomfort.

Cardiac function failure [12]: Grade I: Poor peripheral circulation perfusion, hypotension, and systolic blood pressure below 10.7 kPa for more than one hour. Grade II: Congestive cardiac failure, characterized by paroxysmal nocturnal dyspnea, jugular vein distension; rales at the bases of both lungs; cardiomegaly; acute pulmonary edema; gallop rhythm (first heart sound), and increased venous pressure. The occurrence of any of these conditions was defined as CTRCD. Once CTRCD was confirmed, appropriate treatment was administered based on each patient's condition.

Response Evaluation Criteria in Solid Tumors [13]: Complete Response (CR): complete disappearance of the tumor lesion after chemotherapy for more than 4 weeks; Partial Response (PR): a reduction in lesion size of 30% or more after 4 weeks; Stable Disease (SD): no CR or PR and no increase in lesion size; Progression Disease (PD): a 20% or greater increase in lesion size. Objective response rate (ORR) = (CR + PR)/total number of patients × 100.

Data extraction

All patients underwent echocardiography before chemotherapy (T0), during chemotherapy cycle 3 (T1), and upon completion of chemotherapy (T2) using the E95 GE system (GE

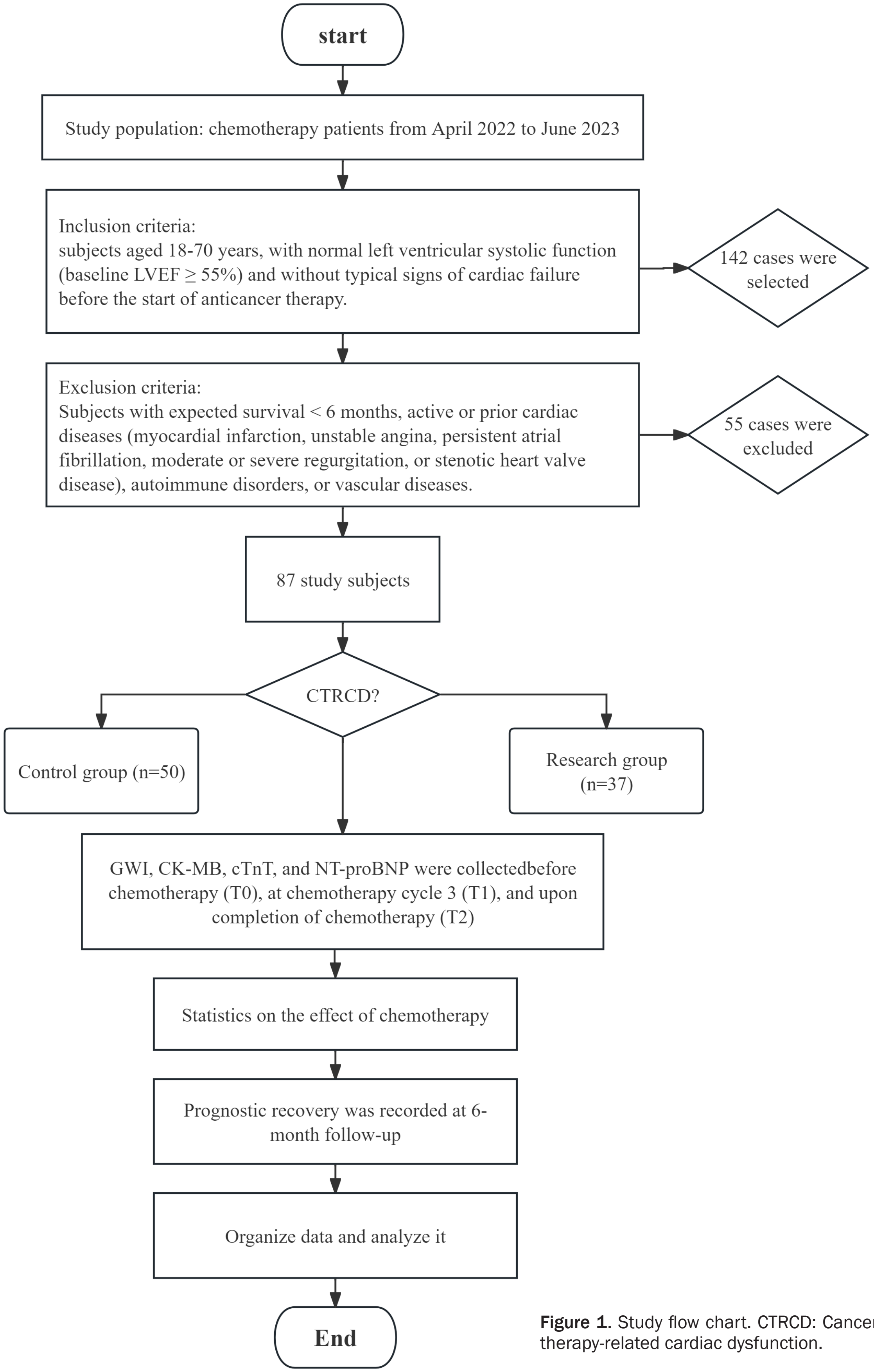


Figure 1. Study flow chart. CTRCD: Cancer therapy-related cardiac dysfunction.

Table 1. Comparison of baseline data between the two groups

Group	n	Age	Course of disease (month)	Male/female	Lung cancer/breast cancer/esophageal cancer/other	BMI (kg/m^2)	Smoking/non-smoking	Alcohol consumption/non-consumption
Control group	50	65.22 ± 3.69	6.26 ± 2.95	30 (60.00)/ 20 (40.00)	21 (42.00)/19 (38.00)/ 8 (16.00)/2 (3.51)	22.16 ± 1.85	37 (74.00)/ 13 (26.00)	19 (38.00)/ 31 (62.00)
Research group	37	65.46 ± 4.19	6.27 ± 3.75	18 (48.65)/ 19 (51.35)	15 (40.54)/16 (43.24)/ 5 (13.51)/1 (2.70)	22.62 ± 1.66	26 (70.27)/ 11 (29.73)	13 (35.14)/ 24
t (χ^2)		0.282	0.014	0.142	0.257	1.182	0.148	0.075
P		0.778	0.989	0.706	0.968	0.240	0.700	0.784

Note: BMI: body mass index.

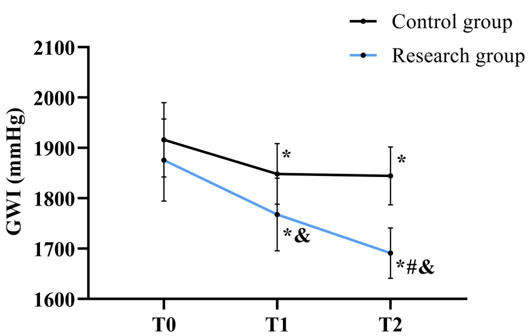


Figure 2. Comparison of myocardial GWI between two groups at T0-T2. T0: before chemotherapy; T1: during chemotherapy cycle 3; T2: upon completion of chemotherapy; GWI: Global work index. **P* < 0.05, compared with T0; #*P* < 0.05, compared with T1; &*P* < 0.05, compared with control group.

Company, USA) equipped with an M5S sensor (3.5 MHz). LVEF and myocardial GWI were recorded. Fasting venous blood samples were collected from patients at T0, T1, and T2 and stored in procoagulant tubes. The samples were allowed to stand at room temperature for 30 min and then centrifuged for serum separation. Subsequently, levels of creatine Kinase-MB (CK-MB), cardiac troponin T (cTnT), and N-terminal pro-brain natriuretic peptide (NT-proBNP) were measured using a multifunctional microplate reader. Patients were followed up for 6 months, with follow-up visits occurring at intervals not exceeding 1 month. Recovery from CTRCD of patients in the research group was recorded. For prognosis, a patient was considered recovered if CTRCD symptoms disappeared; if any symptoms or signs of CTRCD were present, the patient was considered not recovered.

Outcome measures

Primary indicators: Differences in myocardial GWI between the research group and the control group, and the diagnostic performance of myocardial GWI in predicting CTRCD at T0 and T1.

Secondary indicators: (1) Prognosis of recovery from CTRCD in the research group. The relationship between myocardial GWI and the prognosis of CTRCD recovery at T2 was analyzed. (2) Correlation between myocardial GWI and CK-MB and cTnT levels in patients in the research group.

Statistical analysis

Statistical analysis was performed using SPSS 24.0 software. Enumeration data were expressed as [n (%)], and comparisons between groups were performed using the chi-square test. Measurement data were expressed as (Mean ± standard deviation), with comparisons between groups made using the independent sample t-test. Within-group comparisons across multiple time points were analyzed using repeated measures ANOVA and the Bonferroni correction. The predictive value GWI for CTRCD as well as its recovery was assessed using the receiver operating characteristic (ROC) curve, with the area under the curve (AUC) closer to 1 indicating better diagnostic performance. The correlation between variables was analyzed using the Pearson correlation coefficient. Logistic regression was used to identify prognostic factors. A *p*-value of < 0.05 was considered statistically significant.

Results

Comparison of clinical baseline data between the two groups

There was no statistically significant difference in age, gender, and tumor type between the two groups (*P* > 0.05), indicating comparability (**Table 1**).

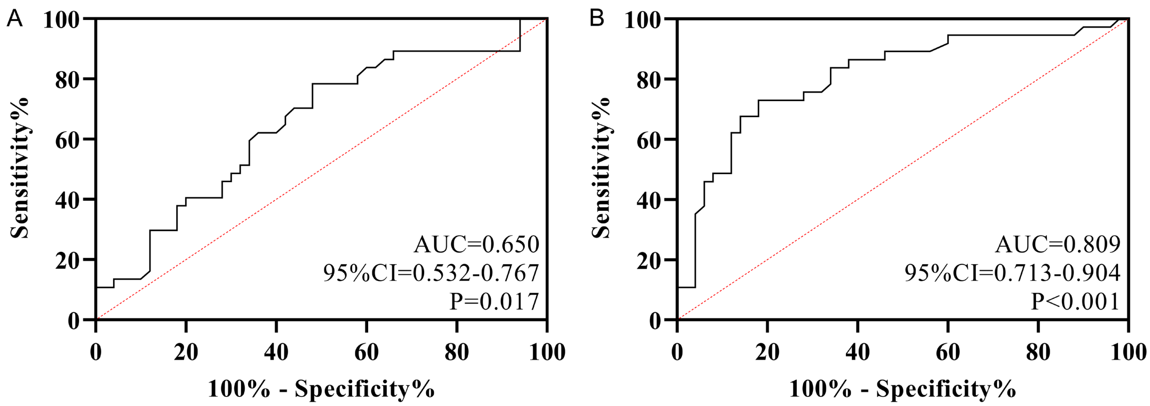


Figure 3. The predictive value of myocardial GWI for CTRCD. A. ROC curve for GWI at T0 in predicting CTRCD. B. ROC curve for GWI at T1 in predicting CTRCD. AUC: Area under curve; ROC: Receiver operating characteristic; CI: Confidence interval (CI). T0: before chemotherapy; T1: during chemotherapy cycle 3. CTRCD: Cancer therapy-related cardiac dysfunction.

Comparison of changes in myocardial GWI between the two groups

At T0, the myocardial GWI in the research group was slightly lower than that in the control group but not reaching statistical significance (P > 0.05). At T1, the myocardial GWI decreased in both groups and was significantly lower in the research group (P < 0.05). In the control group, the myocardial GWI at T2 was comparable to that at T1 but was lower than that at T0 (P < 0.05). In the research group, the myocardial GWI at T2 was lower than both that at T1 and that in the control group (both P < 0.05) (**Figure 2**).

Predictive value of myocardial GWI for CTRCD development during chemotherapy

According to ROC analysis, the sensitivity and specificity of myocardial GWI < 1916 mmHg at T0 for predicting CTRCD were 78.38% and 52.00%, respectively (P < 0.05). The sensitivity and specificity of myocardial GWI < 1804 mmHg at T1 for predicting CTRCD were 72.97% and 82.00%, respectively (P < 0.05) (**Figure 3**).

Comparison of cardiac function between the two groups

Between-group comparison showed that CK-MB, cTnT, and NT-proBNP levels at T0-T2 were significantly higher in the research group compared to the control group (P < 0.05). Within-group comparison revealed that CK-MB at T2 was elevated in the research group compared to T0 (P < 0.05), whereas CK-MB and cTnT levels did not show significant changes at T0-T2 in the control group (P > 0.05) (**Figure 4**).

Relationship between myocardial GWI and cardiac function in patients with CTRCD

Pearson correlation coefficient analysis showed that myocardial GWI at T0, T1, and T2 was negatively correlated with CK-MB, cTnT, and NT-proBNP in patients from the research group (P < 0.05). Specifically, lower myocardial GWI was associated with higher levels of CK-MB, cTnT, and NT-proBNP (**Figure 5**).

Relationship between GWI and chemotherapy effect

As shown in **Table 2**, the ORR of the research group was 54.05%, significantly lower than 76.00% in the control group (P < 0.05, **Table 2**). Further comparison of GWI among patients with different chemotherapy efficacy showed no significant differences (P > 0.05) (**Figure 6**).

Relationship between myocardial GWI and CTRCD prognosis

During the 6-month follow-up, 22 patients in the research group recovered from CTRCD, while the other 15 patients continued to experience varying degrees of CTRCD. Comparison showed that the myocardial GWI at T2 was higher in patients who recovered from CTRCD compared to those who did not (P < 0.05, **Figure 7A**). According to the ROC curve, the

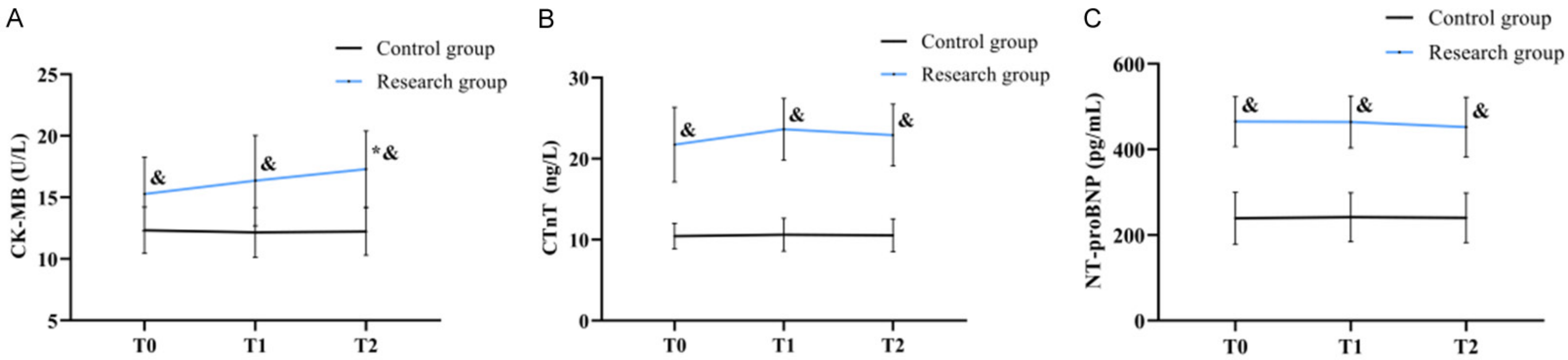
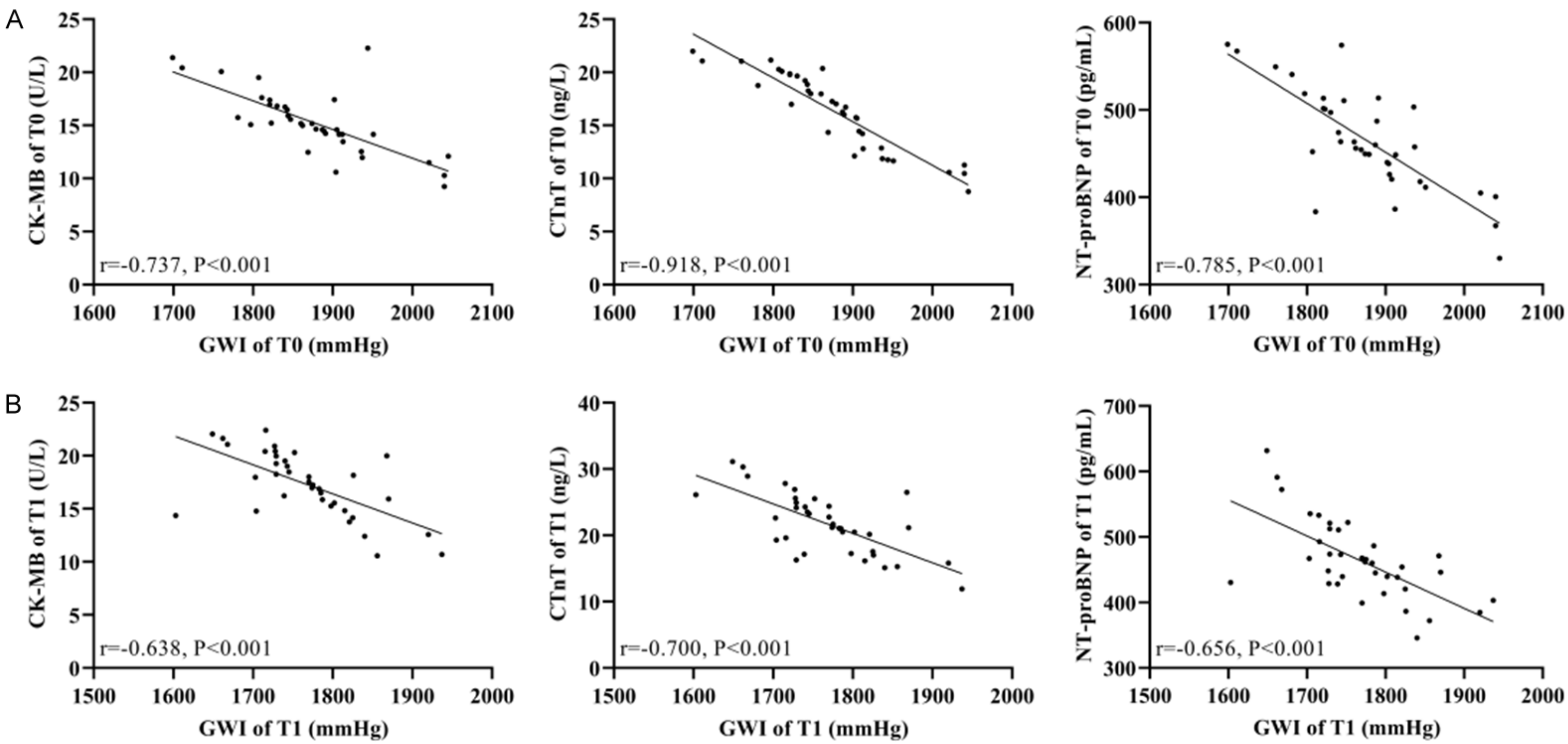


Figure 4. Comparison of cardiac function between the two groups at T0-T2. A. Comparison of CK-MB; B. Comparison of CTnT; C. Comparison of NT-proBNP. *P < 0.05, compared with T0; &P < 0.05, compared with control group. CK-MB: Creatine Kinase-MB; cTnT: Cardiac troponin T; NT-proBNP: N-Terminal Pro-Brain Natriuretic Peptide. T0: Before chemotherapy; T1: during chemotherapy cycle 3; T2: upon completion of chemotherapy.



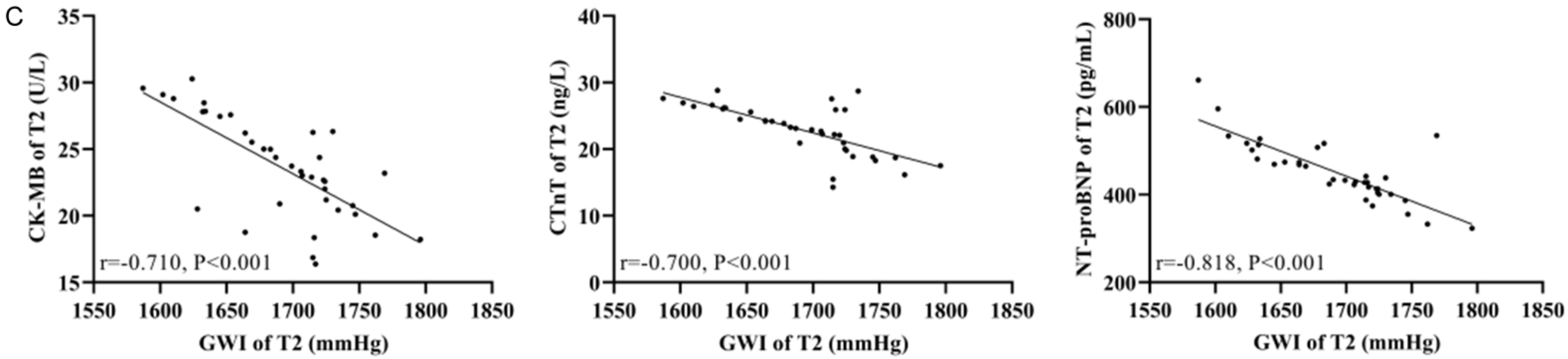


Figure 5. Relationship between myocardial GWI and cardiac function in patients with CTRCD. A. The relationship between CK-MB, cTnT, NT-proBNP with GWI at T0. B. The relationship between CK-MB, cTnT, NT-proBNP with GWI at T1. C. The relationship between CK-MB, cTnT, NT-proBNP with GWI at T2. GWI: Global work index; CK-MB: Creatine Kinase-MB; cTnT: Cardiac troponin T; NT-proBNP: N-Terminal Pro-Brain Natriuretic Peptide. T0: Before chemotherapy; T1: during chemotherapy cycle 3; T2: upon completion of chemotherapy. CTRCD: Cancer therapy-related cardiac dysfunction.

Table 2. Clinical outcomes of the two groups of patients

Group	n	CR	PR	SD	PD	ORR
Control group	50	14 (28.00)	24 (48.00)	8 (16.00)	4 (8.00)	76.00
Research group	37	8 (21.62)	12 (32.43)	12 (32.43)	5 (13.51)	54.05
χ^2						4.609
P						0.032

Note: CR: Complete Response; PR: Partial Response; SD: Stable Disease; PD: Progression Disease; ORR: Objective response rate.

sensitivity and specificity of myocardial GWI > 1716 mmHg at T2 for predicting recovery from CTRCD were 59.09% and 93.33%, respectively ($P > 0.05$) (**Figure 7B**). In addition, we performed logistic regression analysis, with the patients' prognosis (recovered or not) as the dependent variable and GWI as the covariate, revealing that GWI was an independent risk factor for CTRCD recovery (**Table 3**). In addition, we developed a predictive model for the prognostic recovery of patients with CTRCD and found that GWI demonstrated strong prognostic performance ($P < 0.05$, **Figure 8**).

Discussion

Tumors are a major threat to human health and survival, making clinical treatment critically important [14]. Chemotherapy, a primary treatment option for tumors, is necessary for nearly all cancer patients. In individuals with thoracic tumors, CTRCD is a major complication during chemotherapy, impacting recovery and prognosis. Preventing CTRCD has become a key focus in modern clinical research.

In this study, we found that GWI was reduced in patients with CTRCD, consistent with the findings of Chaganti BT et al. [15]. The observed decrease in myocardial GWI at T1 and T2 in both groups was likely associated with the toxic effects of chemotherapy on cardiac function. In the research group, the occurrence of CTRCD led to further deterioration of cardiac functions, resulting in a more pronounced decrease in myocardial GWI. On the other hand, two-dimensional global longitudinal strain (GLS) echocardiography, with its high reproducibility, can detect early myocardial dysfunction, predict CTRCD, and guide cardiac protection. It may serve as a useful alternative to LVEF monitoring in patients [16]. However, Mahdiui ME et al. pointed out that GLS's load dependence during continuous follow-up limits its application, as changes in systolic blood pressure (SBP) may affect its measurement [17]. To address this, Cronin M et al. developed a noninvasive method that combines echocardiographic GLS, SBP, and flow measurements from the aortic and mitral valves to assess left ventricular mass. This method has shown a strong correlation with invasive measurements of myocardial performance and metabolism using positron emission computerized tomography [18]. While studies have confirmed the diagnostic and prognostic value of myocardial GWI in various cardiovascular diseases [19, 20], its application in patients receiving cancer therapy remains unexplored. Based on our findings, myocardial GWI exhibited excellent efficacy in predicting the occurrence of CTRCD during chemotherapy in patients with thoracic tumors, indicating its potential for future clinical application. Specifically, we observed superior efficacy of myocardial GWI in diagnosing CTRCD at T1. This may be attributed to the more remarkable decrease in myocardial GWI in patients already developing CTRCD compared to those without, which enhances the diagnostic sensitivity of myocardial GWI for CTRCD at T1.

In the comparison of clinical efficacy, we found that the ORR of the research group was significantly lower than that of the control group, demonstrating that the occurrence of CTRCD can lead to a reduction in the effectiveness of chemotherapy in patients. Similar results were reported in the study by Avula V et al. [21]. However, no significant difference was seen in the comparison of myocardial GWI between patients with different chemotherapy efficacies, suggesting that GWI has strong specificity and is not affected by tumor progression. This further emphasizes the potential of GWI as a diagnostic tool for CTRCD. Early diagnosis and treatment of CTRCD are critical for improving prognosis. Currently, the diagnosis of CTRCD primarily relies on the reduction of LVEF; however, LVEF measurement is a relatively insensitive tool for detecting cardiotoxicity in the early

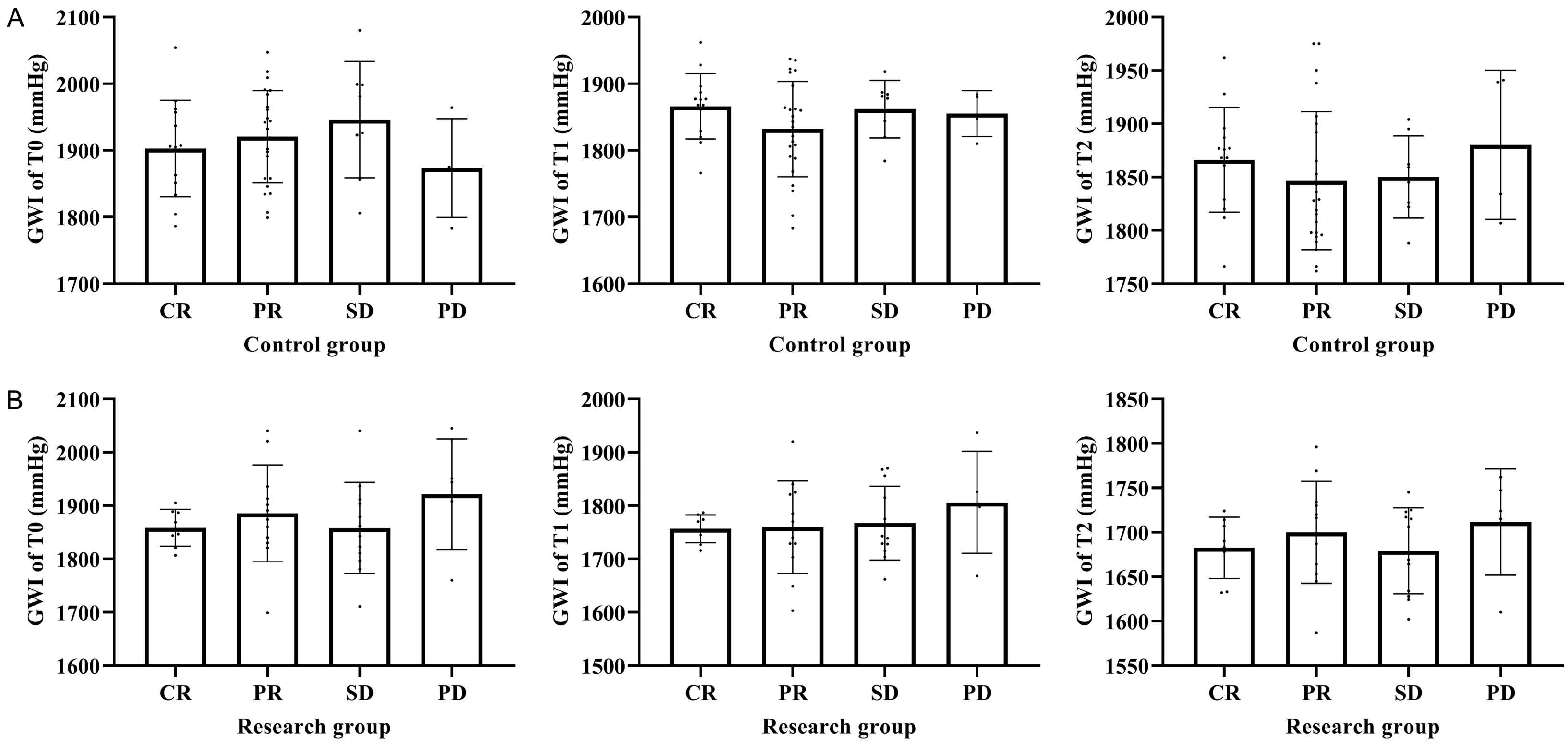


Figure 6. Relationship between GWI and chemotherapy effect. A. Comparison of GWI in patients with different treatment efficacy in the control group. B. Comparison of GWI in patients with different treatment efficacy in the research group. CR: Complete Response; PB: Partial Response; SD: Stable Disease; PD: Progression Disease; GWI: Global work index. T0: Before chemotherapy; T1: during chemotherapy cycle 3; T2: upon completion of chemotherapy.

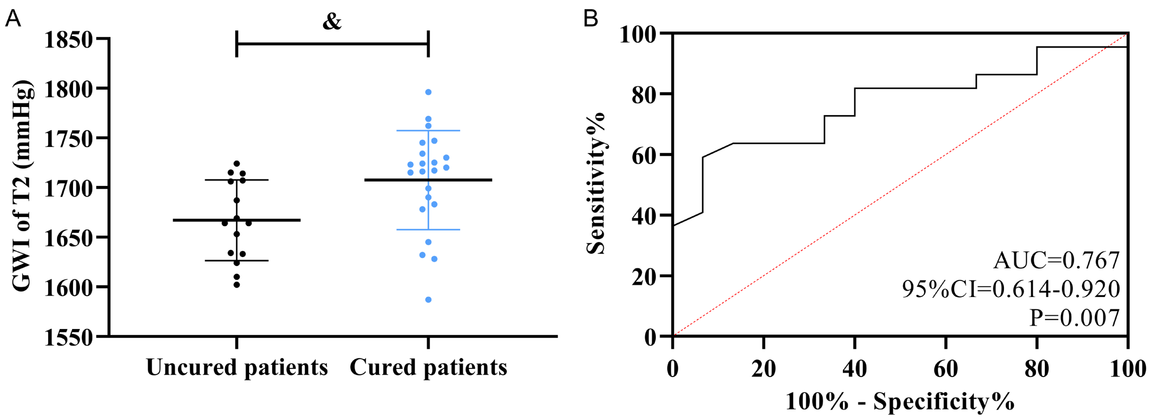


Figure 7. The relationship between GWI and prognosis and rehabilitation of CTRCD. A. Comparison of GWI between rehabilitated and unrehabilitated patients at T2. B. ROC curve for GWI at T2 for predicting recovery from CTRCD. GWI: Global work index; AUC: Area under curve; CI: Confidence interval. T2: upon completion of chemotherapy. CTRCD: Cancer therapy-related cardiac dysfunction.

Table 3. The effect of GWI on prognostic recovery in CTRCD

	β	S.E.	Wald χ^2	P	OR	95% CI
GWI	0.426	0.116	10.264	< 0.001	1.424	1.104-4.269

Note: β: Regression coefficient; S.E.: Standard error; OR: Odds ratio; CI: Confidence interval.

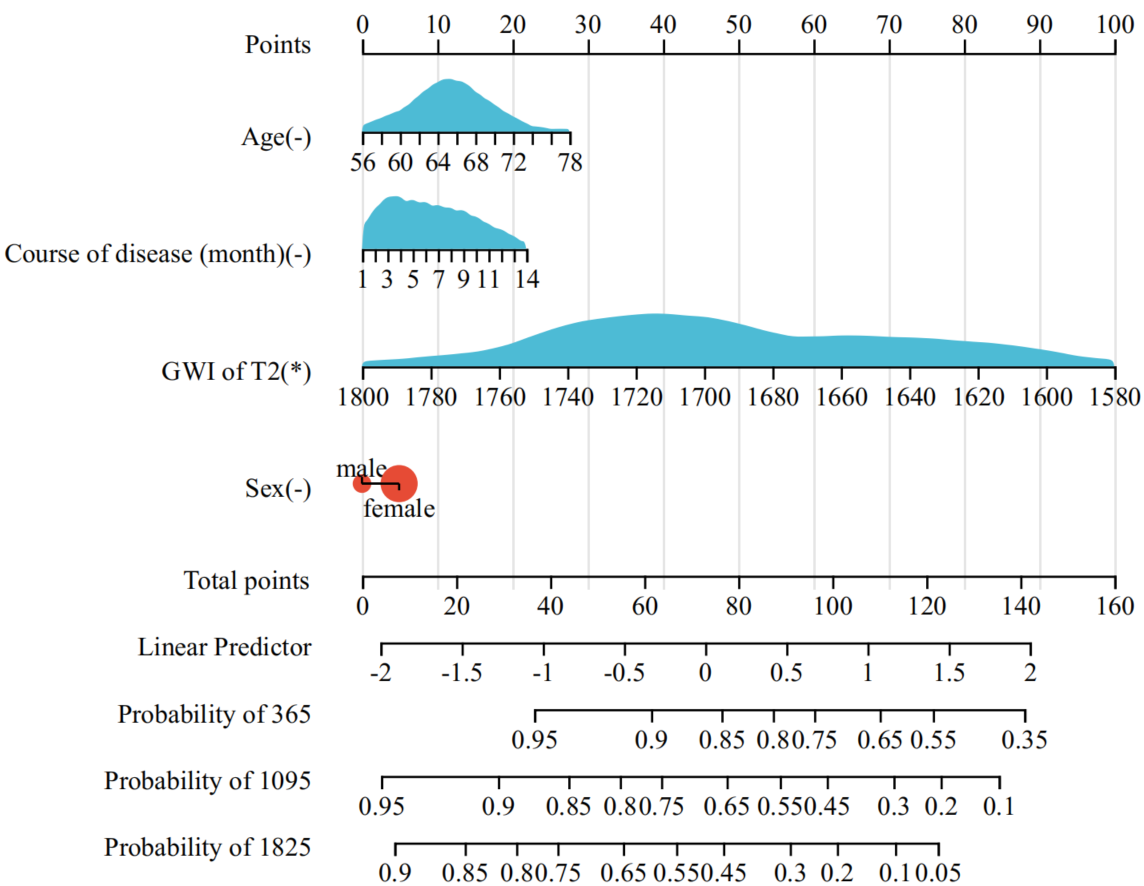


Figure 8. Predictive model for recovery from CTRCD. **P* < 0.05. GWI: Global work index; CTRCD: Cancer therapy-related cardiac dysfunction. T2: upon completion of chemotherapy.

stage, as it may only change obviously once substantial myocardial injury has occurred and compensatory mechanisms are exhausted [22]. In contrast, the use of myocardial GWI greatly compensates for the limitations of LVEF in diagnosis, providing a more reliable method for ensuring cardiac safety in patients undergoing chemotherapy for thoracic tumors. Furthermore, the results of further prognostic follow-up showed that myocardial GWI at T2 also exhibited excellent efficacy in predicting the prognosis of CTRCD recovery, which again corroborates the close relationship between myocardial GWI and CTRCD. Meanwhile, logistic regression analysis and predictive modeling also indicated that GWI was an independent factor affecting the prognosis of recovery in patients with CTRCD. These results suggest that myocardial GWI levels can be used for early assessment of CTRCD prognosis and recovery in future clinical practice, enabling earlier implementation of targeted interventions.

In the comparison of cardiac function, we also observed that CK-MB, cTnT, and NT-proBNP levels in the research group were significantly higher than those in the control group. The study by Islam O et al. mentioned that the typical clinical presentation of CTRCD includes elevated CK-MB, cTnT, and NT-proBNP levels [23], and our findings align with these views. Through correlation analysis, we found that myocardial GWI at T0, T1, and T2 was negatively correlated with CK-MB, cTnT, and NT-proBNP in the research group, further validating the close relationship between myocardial GWI and cardiac function in patients with CTRCD. Consistent with our findings, Lakatos BK et al. observed a negative correlation between myocardial GWI and CK-MB in patients with cardiac failure [24], suggesting that myocardial GWI may not only be useful for assessing CTRCD but also serve as a monitoring parameter for cardiac function, providing patients with more reliable safety assurances.

Due to the small sample size, the findings of this study exhibit a certain degree of randomness. Additionally, only patients with thoracic tumors were included, so it remains unclear whether myocardial GWI is still effective in predicting CTRCD associated with tumors in other body regions. These limitations highlight the need for further validation with a larger number of cases. In the future, more in-depth and comprehensive analyses of the relationship between myocardial GWI and CTRCD in tumor patients will be necessary to provide more reliable clinical references.

Conclusion

Myocardial GWI demonstrates excellent efficacy in predicting CTRCD during chemotherapy for patients with thoracic tumors. By monitoring changes in myocardial GWI, the occurrence of CTRCD can be detected at an early stage, enabling timely intervention and providing patient safety during chemotherapy, ultimately enhancing their prognosis.

Acknowledgements

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Disclosure of conflict of interest

None.

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