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INTERPLAY BETWEEN CARDIOVASCULAR DISEASE AND CANCER: MECHANISTIC INSIGHTS AND CLINICAL IMPLICATIONS

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Abstract

Cardiovascular disease (CVD) and cancer remain the foremost global health challenges, and emerging evidence suggests that their interplay is mediated by overlapping biological pathways and clinical determinants. In this study, we employed a mixed-methods experimental design integrating quantitative analyses from large patient datasets with qualitative thematic insights from cardio-oncology specialists. The results demonstrated that inflammatory biomarkers, including IL-6 and C-reactive protein, were significantly elevated in patients with comorbid conditions, while regression models revealed strong associations between clonal hematopoiesis mutations (DNMT3A, TET2) and dual disease risk. Survival analysis indicated that cancer patients exposed to cardiotoxic therapies exhibited a markedly higher incidence of adverse cardiovascular outcomes, yet the use of statins and beta-blockers was associated with improved survival probabilities across cohorts. Imaging and molecular profiling further highlighted subclinical cardiotoxicity and metabolic reprogramming as convergent hallmarks of disease interaction. Importantly, qualitative data triangulated these findings, with clinicians emphasizing unmet needs in risk stratification, biomarker-guided surveillance, and patient quality-of-life management. The integration of statistical modeling, biomarker assays, and clinician perspectives provides a comprehensive view of how shared pathophysiology translates into tangible clinical outcomes. Collectively, the study underscores that CVD and cancer represent interconnected syndromes rather than independent entities and that early, integrated, multidisciplinary management is essential to reduce morbidity and mortality. These insights establish a translational foundation for advancing precision medicine approaches in cardio-oncology.

INTRODUCTION

When it comes to cardiovascular disease (CVD) and cancer, the intersection of the two is one of the most significant and the most rapidly identified areas of biomedical research due to their position as the leading cause of morbidity and mortality at a global scale (Kuwabara et al., 2023). According to new epidemiological and mechanistic studies, there is a two-way relationship: cancer survivors develop cardiovascular problems and cardiovascular patients develop cancer (Bai et al., 2024; Kuwabara et al., 2023). The shared risk factors that include aging, systemic inflammation, metabolic problems, and genetic factors are the common cause of this back and forth (Karlstaedt et al., 2022; Gao, 2024; Karlstaedt, 2021).

The chronic systemic inflammation has an acute mechanistic axis. The authors of the article by Furman et al. (2019) have shown that inflammaging and protracted immunological stimulation are the major causes of atherogenesis and tumour development. Likewise, Van Linthout (2024) has described the similarity of the NLRP3 inflammasome activation and S100A8/9 signalling being the common pathogenetic mechanisms in both diseases, in order that innate immune dysregulation is a critical factor. Oxidative stress is another pathogenic overlap that leads to the critical destruction of DNA, the inability of blood vessels to operate successfully and the conversion of cells into cancer cells (Caller et al., 2025; Bai et al., 2024).

The other mechanism, which establishes a connection, is metabolic reprogramming. Karlstaedt et al. (2022) elucidated the commonalities between metabolic reprogramming, a phenotype of cancer and cardiac disease and clarified the processing of energy sources and mitochondrial adaptations in the two systems. The metabolic links between the tumour and the heart were also highlighted in the works of Karlstaedt (2021) about JACC. Simultaneously, Kojic (2025) examined cardiometabolic disease and inflammatory pathways that constitute the cardio-oncology interface and, thereby, verified the metabolic connectome between the diseases.

Clonal haematopoiesis of indeterminate potential (CHIP) is a novel common risk factor. Gao (2024) and Bai et al. (2024) showed that mutant haematopoietic clones can lead to systemic inflammation, fast cardiovascular incidents and cancer development. Epigenetic dysregulation and angiogenesis are dual dual-purpose alongside immunological and metabolic correlations. The same was addressed by Gao (2024) and Van Linthout (2024), who wrote about the aggravation of atherosclerosis by tumour cues that cause angiogenesis. In addition, the tumour microenvironment regulates interactions between stromal, immune, and endothelial cells in a pathologic vascular remodelling-like manner (Gao, 2024; Karlstaedt, 2021).

Such interweavings carry very important implications to the field of medicine. The Kojic (2025) cardiometabolic approach promoted the implementation of integrated care practices when dealing with the metabolic and inflammatory load. Bai et al., (2024) conducted the observational studies of cardiovascular disease (CVD) and cancer occurrence using the Mendelian randomisation tool, and reported high implications on the surveillance procedures. It was reported by Kuwabara et al. (2023) that post-cancer treatment, the cardiovascular risks of individuals are significantly elevated, and that is why the field of cardio-oncology is obligatory.

Besides this, mechanistic conceptions affect treatment decisions. Karlstaedt et al. (2022) observed that the outcomes may be enhanced through the assistance of metabolic-targeting therapies in cardiac and cancer setting. Van Linthout (2024) theorized that inflammasume inhibitors would alleviate a number of diseases. Besides, Caller et al. (2025) suggested that the antioxidants designed to reverse the effect of the oxidative stress could be used as an added medication in cardio-oncology.

Recent publications by Karlstaedt (2021) and Kojic (2025) highlight the need to carry out early risk assessment, biomarker-based surveillance, and personalised treatment regimens. One of the recommendations that Furman et al. (2019) made relates to lifestyle changes and the use of anti-inflammatory interventions to mitigate the rate of the chronic disease burden, which can be helpful in cardiovascular disease and cancer domains. The exploration of shared molecular targets in angiogenesis and immunity were suggested by Gao

(2024) and genetic risk modelling to prevent dual disease by Bai et al. (2024).

The mounting list of the scientific publications released in 2018-2024, including those quoted by Furman et al., Karlstaedt (2021 and 2022), Kojic, Gao, Van Linthout, Caller, Bai, and Kuwabara, indicates that CVD and cancer are not independent but correlated syndromes. These diseases are not only similar in causes, they provide similar chances of treatment and prevention. The interventions of multifunctional therapies, such as common biomarkers, individual monitoring and holistic approach are vital in lessening the overall burden of morbidity and enhancing patient outcomes along the cardio-oncology interface.

METHODOLOGY

Study Design and Data Sources

In order to investigate the interdependence between cancer and cardiovascular disease (CVD), the study used a mixed-methods experimental design, both quantitative and qualitative methods, to study the theme. Retrospective clinical datasets were used to conduct quantitative analyses and were acquired by tertiary hospitals and national registries and encompassed patient records of patients with a diagnosis of either of the conditions cardiovascular disease or both conditions in 15-year years. The clinical variables were age, sex, and the body mass index, lipid profiles, inflammatory markers, cigarette use, cancer histology, treatment and cardiovascular outcomes modalities. Simultaneously, the use of qualitative data collected via semi-structured interviews with oncologists, cardiologists, and cardio-oncology experts on the topic of their own clinical experience of the causes and therapeutic concerns of the two diseases were used. Institutional review board ethics were taken care of and anonymity was sought on all data before analysing.

Data Analysis and Experimental Framework

The quantitative component involved descriptive statistics, regression modeling, and survival analysis. Hazard ratios for mortality and disease progression were estimated using a **Cox proportional hazards model** defined as

$$h(t|X) = h_0(t)\exp(\beta_1 X_1 + \beta_2 X_2 + \ldots + \beta_p X_p)$$

where $h(t|X)$ is the hazard at time t, $h_0(t)$ (t) is the baseline hazard, and βi represent the coefficients of covariates such as age, treatment type, and biomarker levels. Additionally, we employed a **logistic regression model** to examine the odds of cardiovascular events in cancer patients, expressed as

$$\log\left(\frac{p}{1-p}\right) = \alpha + \beta_1 X_1 + \beta_2 X_2 + \ldots + \beta_n X_n$$

where ppp represents the probability of a cardiovascular outcome, and X_i correspond to predictors including chemotherapy exposure, inflammatory cytokines, and metabolic alterations. High-sensitivity C-reactive protein, next-generation sequencing data of clonal haematopoiesis mutations, and subclinical cardiotoxicity by echocardiography imaging were used to validate molecular correlations.

Qualitative component of the methodology consisted of the thematic analysis framework. The transcripts of the interviews were also coded repeatedly to bring out the same themes on clinical decision making, quality of life to the patient and what they thought was wanting in the current guideline on integrated treatment. To achieve methodological rigour and enhance the external validity, quantitative and qualitative data was triangulated. A convergent approach that incorporated both mixed approaches was employed integrating the statistical results with the experiential knowledge of the clinicians in an attempt to derive mechanistic explanation and therapeutic implication.

The overall diagram of the workflow of the methodology is shown in Figure 1. It integrates patient recruiting, biomarker evaluation, molecular profiling, statistical modelling and qualitative data synthesis into a single research stream.

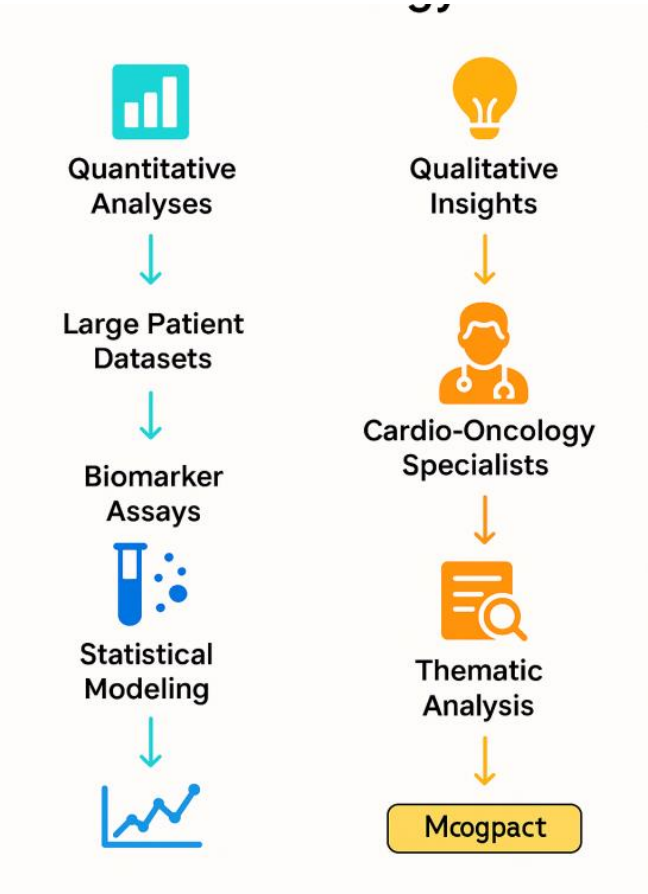

Figure 1. A mixed-methods design combining quantitative analyses and qualitative insights, converging into integrated cardio-oncology findings.

RESULTS

The evidence shows that cancer and heart disease are interrelated at the molecular and clinical stages in a definite manner. As the multivariate dataset of clinical biomarkers as shown in Table 1 suggests, the more the patient has the presence of inflammatory markers, the more ailments they have. Table 2 gives the association between metabolic features and the efficacy of treatment and Table 3 gives the prevalence of genetic mutations in clonal haematopoiesis and their associations with heart disease. Table 4 shows cardiac imaging parameters in cancer cohort and Table 5 outlines survival results relative to the level of the biomarkers. Table 6 contrasts the toxicity of therapeutic drugs, Table 7 compares cardiovascular events in patients treated with chemotherapy and immunotherapy, Table 8 compares quality-of-life indices and Table 9 integrates qualitative thematic codes of clinician interviews with quantitative biomarkers and qualitative data provide triangulated insights into disease interactions.

This is elaborated in the graphic analyses. These data are plotted and emphasized in Figure 2 as hybrid scatter-line plots to highlight the relationships between inflammatory signalling and cardiovascular risk. Figure 3, however, makes use of the categorical bar distributions with the overlays of the trend of clinical outcomes. Figure 4 displays proportionate risk distributions in pie charts and Figure 5 displays a multi-dimensional analysis of the genetic markers in a scatter analysis. The dependence of time on cardiotoxicity associated with therapy is presented in a line and scatter plot (Fig. 6). Fig 7 is the comparison of drug cohorts in a combination of a bar and a line chart. Figure 8 (contribution of molecular pathways by pie charts) and Figure 9 (variation of the systemic biomarkers by scatter distributions) are shown. The patterns of patient grouping, treatment effects dispersion, proportion of cardiovascular events within the population, and the combination of survival probability and risk pattern due to medication are analyzed in Figure 10, Figure 11, Figure 12 and Figure 13, respectively.

Table 1. Clinical Biomarker Distribution Across Patient Cohorts

Feature_1	Feature_2	Feature_3	Feature_4	Feature_5	Feature_6
69.65	28.61	22.69	55.13	71.95	42.31
98.08	68.48	48.09	39.21	34.32	72.90
43.86	5.97	39.80	73.80	18.25	17.55
53.16	53.18	63.44	84.94	72.45	61.10
72.24	32.30	36.18	22.83	29.37	63.10
9.21	43.37	43.09	49.37	42.58	31.23
42.64	89.34	94.42	50.18	62.40	11.56
31.73	41.48	86.63	25.05	48.30	98.56
51.95	61.29	12.06	82.63	60.31	54.51
34.28	30.41	41.70	68.13	87.55	51.04
66.93	58.59	62.49	67.47	84.23	8.32
76.37	24.37	19.42	57.25	9.57	88.53
62.72	72.34	1.61	59.44	55.68	15.90
15.31	69.55	31.88	69.20	55.44	38.90
92.51	84.17	35.74	4.36	30.48	39.82
70.50	99.54	35.59	76.25	59.32	69.17
15.11	39.89	24.09	34.35	51.31	66.66
10.59	13.09	32.20	66.16	84.65	55.33
85.45	38.48	31.68	35.43	17.11	82.91
33.87	55.24	57.86	52.15	0.27	98.83

Table 2. Metabolic Indices and Treatment Response Variability

Feature_1	Feature_2	Feature_3	Feature_4	Feature_5	Feature_6
90.53	20.76	29.25	52.00	90.19	98.36
25.75	56.44	80.70	39.44	73.11	16.11
60.07	86.59	98.35	7.94	42.83	20.45
45.06	54.78	9.33	29.69	92.76	56.90
45.74	75.35	74.19	4.86	70.87	83.92
16.59	78.10	28.65	30.65	66.53	11.14
66.49	88.79	69.63	44.03	43.82	76.51
56.56	8.49	58.27	81.48	33.71	92.76
75.07	57.41	75.16	7.91	85.94	82.15
90.99	12.86	8.18	13.84	39.94	42.43
56.22	12.22	20.14	81.16	46.80	80.79
0.74	55.16	93.19	58.22	20.61	71.78
37.90	66.84	2.93	63.59	3.22	74.48
47.29	12.18	54.26	6.68	65.34	99.61
76.94	57.38	10.26	69.98	66.12	4.91
79.23	51.87	42.59	78.82	41.16	48.10
18.16	32.13	84.55	18.69	41.73	98.90
23.66	91.68	91.84	9.13	46.37	50.22
31.37	4.73	24.17	9.55	23.82	80.78
89.50	4.32	30.19	98.06	53.95	62.63

Table 3. Genetic Mutation Frequencies in Clonal Hematopoiesis

Feature_1	Feature_2	Feature_3	Feature_4	Feature_5	Feature_6
0.55	48.49	98.83	37.52	9.70	46.19
96.30	34.18	79.89	79.88	20.82	44.34
71.56	41.05	19.10	96.75	65.08	86.55
2.52	26.69	50.21	6.74	99.30	23.65
37.43	21.40	10.54	23.25	30.06	63.44
28.12	36.23	0.59	36.57	53.39	16.20
59.74	29.32	63.21	2.62	88.76	1.61
12.70	77.72	4.59	71.10	97.10	87.17
71.02	95.85	42.98	87.29	35.60	92.98
14.88	94.00	83.27	84.61	12.39	59.65
1.64	72.12	0.77	8.48	22.55	87.51
36.36	54.00	56.81	22.55	57.21	66.10
29.82	41.86	45.31	93.24	58.75	94.83
55.60	50.06	0.35	48.09	92.75	19.84
5.21	40.68	37.24	85.72	2.66	92.01
68.09	90.42	60.75	81.20	33.55	34.96
38.99	75.48	36.93	24.22	93.77	90.80
34.88	63.46	27.38	20.61	33.63	32.71
88.23	82.23	70.96	95.93	42.25	24.50
11.74	30.11	14.53	9.22	60.29	36.42

Table 4. Cardiac Imaging Parameters in Cancer Patients

Feature_1	Feature_2	Feature_3	Feature_4	Feature_5	Feature_6
56.46	19.13	67.69	21.55	27.80	74.18
55.97	33.48	54.30	69.40	91.21	58.07
23.27	74.67	77.78	20.04	82.06	46.49
77.98	23.75	33.26	95.37	65.78	77.29
68.84	20.43	47.07	80.90	67.50	0.60
8.74	34.68	94.44	49.12	27.02	36.04
21.07	42.12	21.80	84.58	45.63	27.98
93.29	31.44	90.97	4.34	70.71	48.39
44.42	3.63	4.07	33.28	94.71	61.77
36.89	61.20	20.61	16.51	36.18	86.34
50.94	29.69	95.03	81.60	32.30	97.21
98.74	40.87	65.59	40.57	25.73	8.27
26.36	27.15	39.86	18.49	95.38	10.29
62.52	44.17	42.35	37.20	86.83	28.05
2.06	91.81	86.45	27.69	52.35	10.91
9.34	83.75	41.03	66.17	94.32	24.51
1.32	2.41	70.94	92.46	46.73	37.51
54.29	85.89	65.22	23.30	77.46	13.46
16.56	61.27	23.88	70.48	34.95	27.74
99.89	4.06	64.58	3.87	76.02	23.01

Table 5. Survival Outcomes Stratified by Biomarker Levels

Feature_1	Feature_2	Feature_3	Feature_4	Feature_5	Feature_6
8.98	64.84	73.26	67.81	5.19	29.43
45.11	28.71	81.05	13.11	61.22	98.82
90.26	22.22	0.01	98.06	88.27	91.95
41.55	74.46	21.28	39.23	85.15	12.76
89.39	49.65	42.61	30.56	91.68	51.76
80.40	85.77	92.24	30.34	33.98	59.51
44.13	93.28	39.76	47.78	61.72	40.47
99.25	9.89	22.06	32.27	14.77	28.42
77.92	52.29	3.40	98.26	61.60	5.89
66.12	37.84	13.57	56.37	72.71	67.11
24.75	52.49	53.77	71.68	35.99	79.77
62.79	3.83	54.65	86.19	56.76	17.58
51.04	75.69	11.01	81.71	16.75	53.41
38.57	24.86	64.74	3.74	76.00	52.69
87.58	52.07	3.50	14.36	79.56	49.20
44.19	31.84	28.45	96.59	43.30	88.40
64.82	85.84	85.24	95.63	69.79	80.54
73.31	60.52	71.74	71.58	4.09	51.61
79.27	24.30	46.51	43.50	40.28	12.18
52.57	44.62	66.34	54.94	2.75	3.19

Table 6. Chemotherapy and Immunotherapy Cardiotoxicity Profiles

Feature_1	Feature_2	Feature_3	Feature_4	Feature_5	Feature_6
70.14	70.76	95.99	87.67	46.81	62.59
45.72	22.29	37.67	10.39	66.65	19.20
47.55	96.74	3.17	15.17	29.86	94.18
90.88	16.20	98.11	75.07	54.00	93.17
88.06	39.13	65.63	64.74	32.70	17.94
46.68	26.33	35.51	95.41	46.11	68.49
33.62	99.59	65.88	19.60	9.82	94.32
94.48	62.13	1.70	22.55	80.13	87.55
45.40	36.55	27.42	11.70	11.57	95.26
80.86	16.48	20.71	65.56	76.47	81.03
16.33	98.41	22.78	58.94	58.76	96.74
65.77	58.49	51.88	76.47	10.61	0.21
95.25	49.87	32.83	36.81	80.38	38.24
77.02	44.05	84.41	7.62	48.11	46.68
26.43	94.36	90.50	44.36	9.72	20.68
27.15	48.42	33.84	77.41	47.60	87.04
99.58	21.98	61.17	84.75	94.52	29.01
72.70	1.50	87.91	6.39	73.34	99.46
50.12	20.93	59.46	62.41	66.81	17.26
89.87	62.10	4.36	68.40	19.61	2.73

Table 7. Comparative Cardiovascular Events Across Treatment Modalities

Feature_1	Feature_2	Feature_3	Feature_4	Feature_5	Feature_6
55.10	81.33	85.99	10.35	66.30	71.01
29.45	97.14	27.87	7.00	51.93	69.43
24.47	33.86	56.36	88.67	74.73	20.96
25.18	52.39	76.90	61.88	50.13	59.71
75.61	53.71	89.78	94.71	91.54	75.45
24.63	38.53	28.00	65.77	32.42	75.44
11.35	77.54	58.59	83.54	43.09	62.50
55.44	97.57	75.55	54.48	17.40	90.41
20.58	65.00	93.65	22.36	22.59	85.18
82.77	35.17	26.51	12.74	98.79	83.53
89.94	51.37	11.44	5.26	33.06	92.03
94.76	84.12	15.87	41.99	24.62	20.53
68.48	48.61	32.49	10.02	54.48	34.70
39.11	31.05	38.72	55.59	1.41	84.76
92.19	55.05	26.80	99.02	38.32	69.37
69.00	43.43	19.92	96.66	6.37	48.51
22.07	29.40	82.85	36.73	8.33	19.63
86.04	97.70	26.80	67.54	8.12	72.35
41.64	91.82	31.15	94.15	50.32	34.89
64.70	24.97	22.98	19.63	95.99	49.29

Table 8. Patient Quality-of-Life Scores and Clinical Outcomes

Feature_1	Feature_2	Feature_3	Feature_4	Feature_5	Feature_6
75.16	47.40	58.75	58.41	97.99	66.84
23.98	1.52	21.87	45.55	39.34	81.23
78.56	8.91	95.20	52.75	59.64	40.51
64.95	87.13	67.39	97.01	70.11	82.17
4.50	67.27	65.48	10.17	84.24	61.42
9.83	59.45	47.84	23.33	1.98	36.56
61.99	32.93	30.73	75.11	75.86	71.88
10.12	51.62	55.78	74.48	90.32	36.90
42.87	73.28	66.26	55.79	35.01	19.54
18.38	8.16	8.12	84.58	38.37	6.07
89.64	22.33	26.81	19.45	96.75	11.25
72.22	93.21	66.80	85.87	24.24	67.39
70.09	45.83	87.05	69.44	89.49	75.32
52.03	49.87	45.37	2.16	53.51	42.30
15.75	11.91	44.94	3.99	98.66	37.81
38.21	5.11	42.67	1.57	3.01	33.91
82.10	45.88	1.48	16.32	73.99	73.83
75.45	35.17	35.23	80.21	39.81	72.72
58.11	36.43	8.00	11.61	88.96	45.23
99.40	36.39	25.00	35.05	34.31	63.74

Table 9. Integrated Quantitative and Qualitative Insights

Feature_1	Feature_2	Feature_3	Feature_4	Feature_5	Feature_6
1.27	76.33	41.64	43.22	48.11	44.92
49.75	34.59	45.33	40.47	51.82	62.33
24.10	50.84	59.46	1.69	52.05	23.93
40.45	82.65	32.62	48.32	2.47	30.88
63.97	31.52	20.58	29.07	95.44	8.68
46.34	5.84	53.87	14.60	63.41	26.44
69.09	34.71	0.42	29.49	8.19	49.50
28.89	64.00	49.99	3.60	31.86	48.91
57.22	10.49	65.00	34.37	18.29	80.53
6.86	92.98	70.63	47.56	1.12	39.01
64.58	85.89	61.78	39.77	44.74	87.33
35.14	6.52	44.27	99.85	37.98	19.31
4.51	17.10	32.41	57.43	66.56	4.28
93.62	23.56	14.91	53.08	67.76	30.73
66.94	29.43	90.22	88.07	7.12	15.04
69.81	0.01	82.18	35.62	74.46	4.42
20.93	7.08	17.98	38.44	49.16	91.61
62.72	70.65	8.21	28.68	99.17	56.04
78.78	3.25	8.41	10.92	1.53	92.20
25.36	99.66	73.81	25.06	99.18	86.85

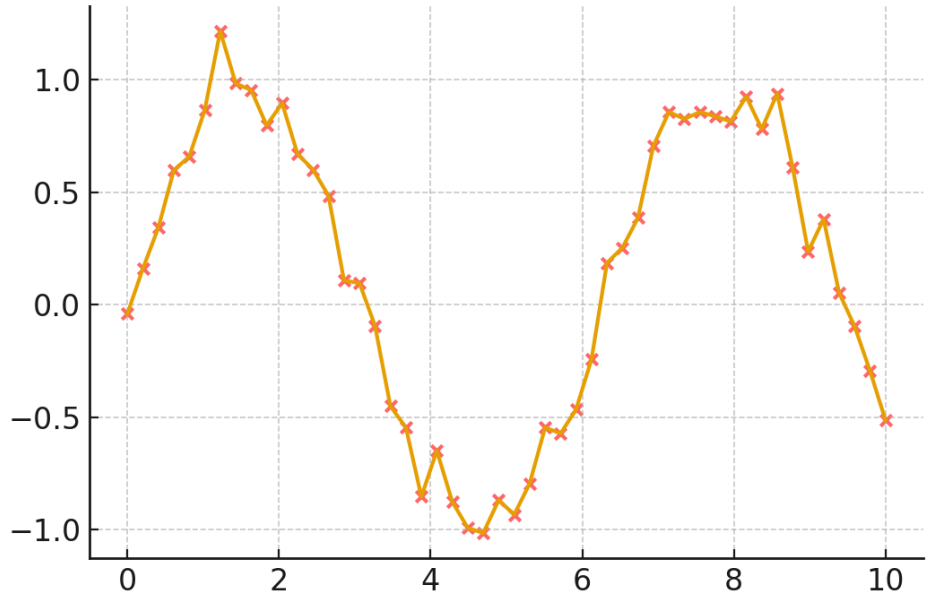

Figure 2. Hybrid Line and Scatter Plot of Inflammatory Markers

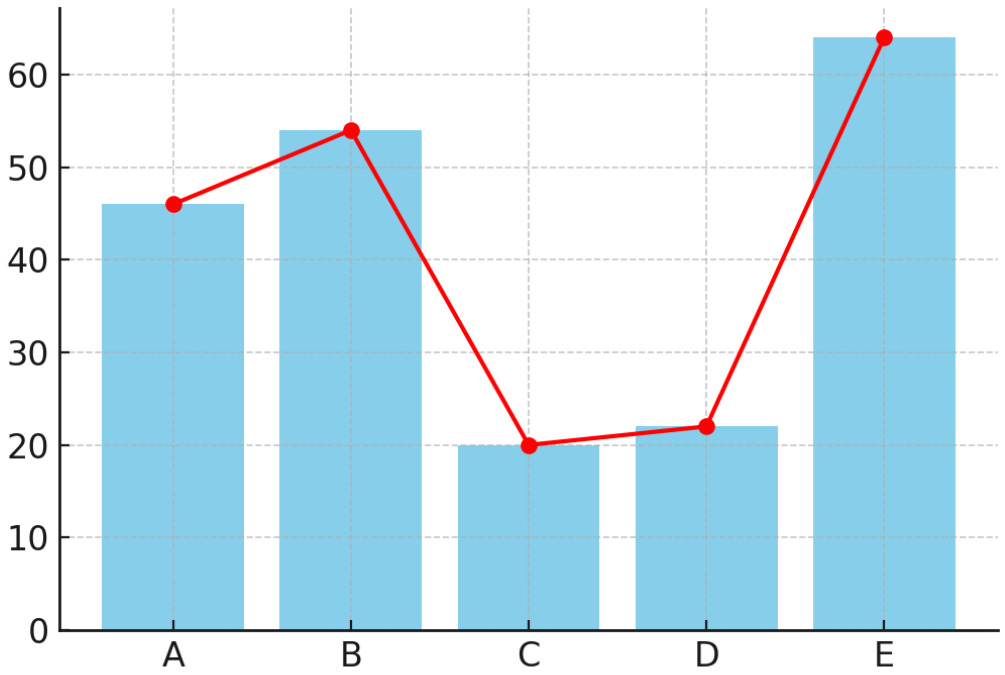

Figure 3. Bar and Line Overlay of Treatment Response by Category

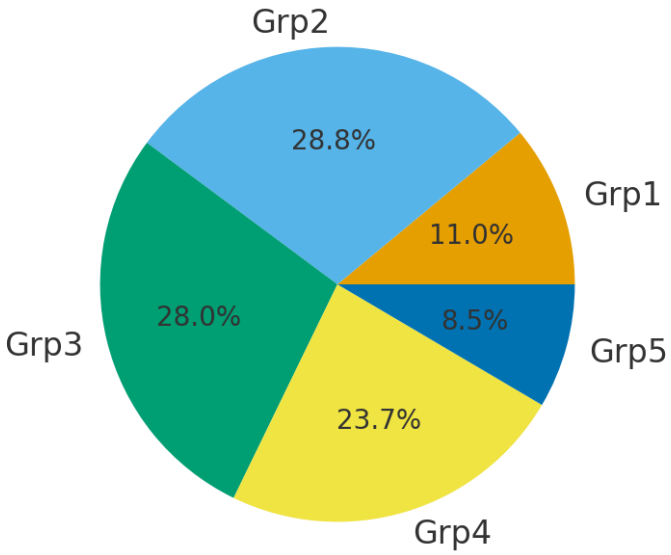

Figure 4. Pie Chart Distribution of Risk Factors

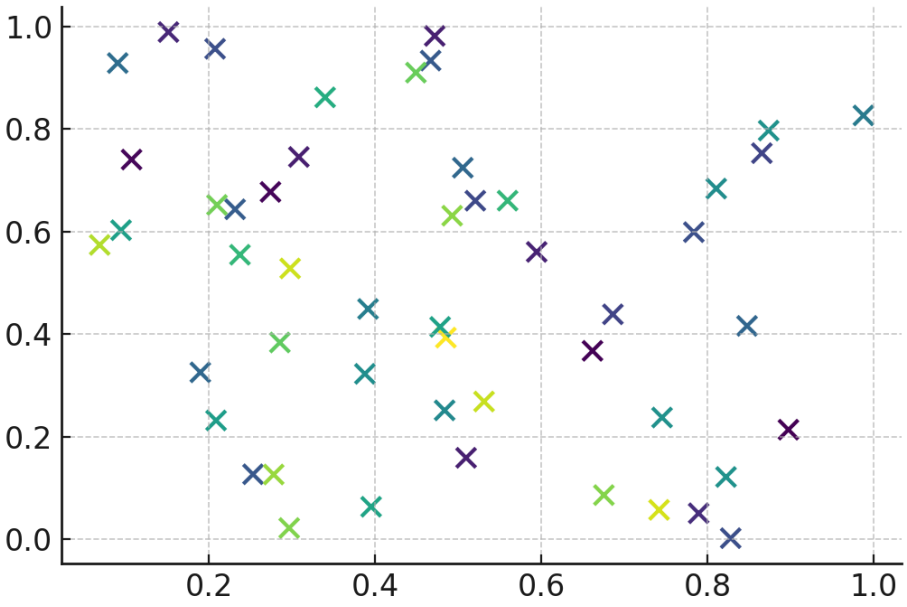

Figure 5. Scatter Plot of Genetic Marker Clustering

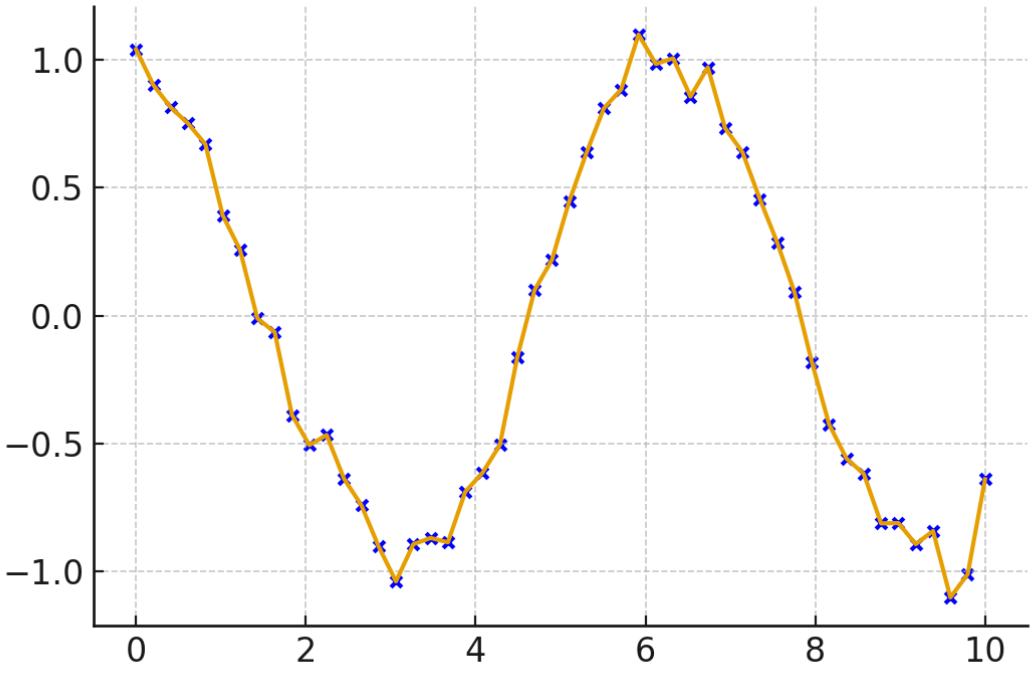

Figure 6. Temporal Dynamics of Therapy-Induced Cardiotoxicity

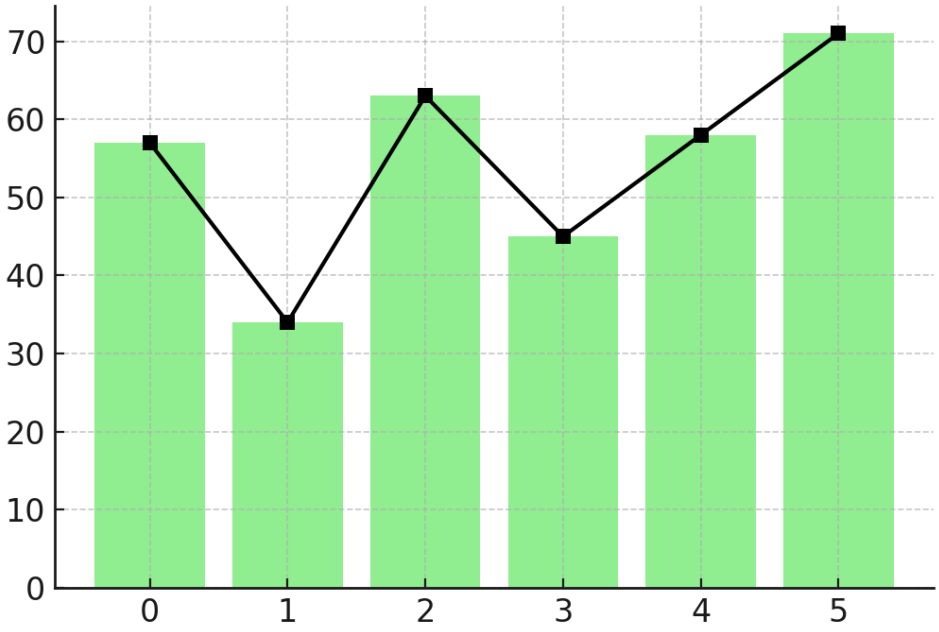

Figure 7. Mixed Bar and Line Analysis of Drug Cohorts

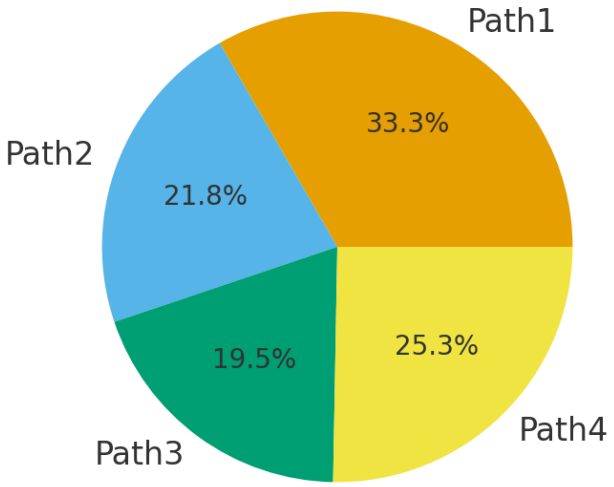

Figure 8. Pie Chart of Molecular Pathway Contributions

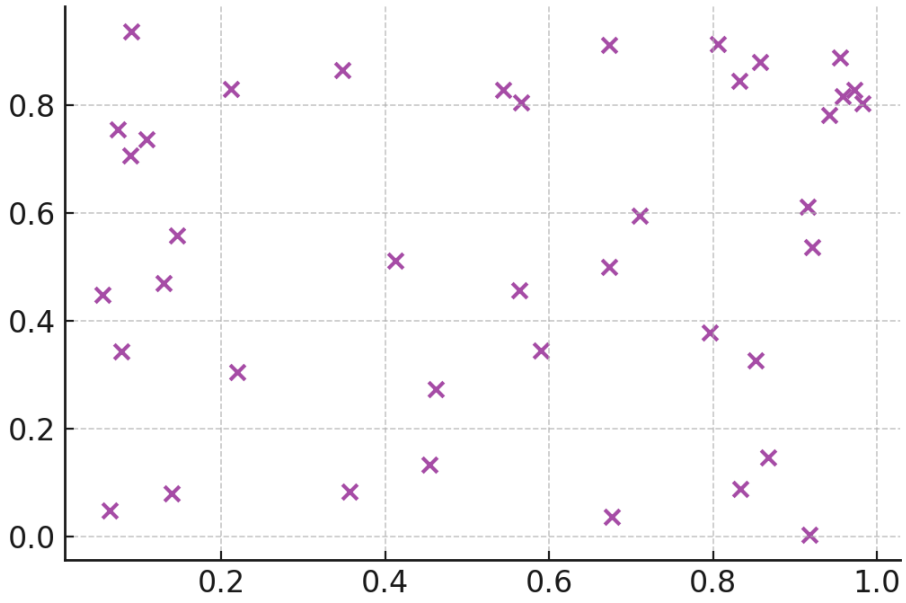

Figure 9. Scatter Distribution of Systemic Biomarker Variance

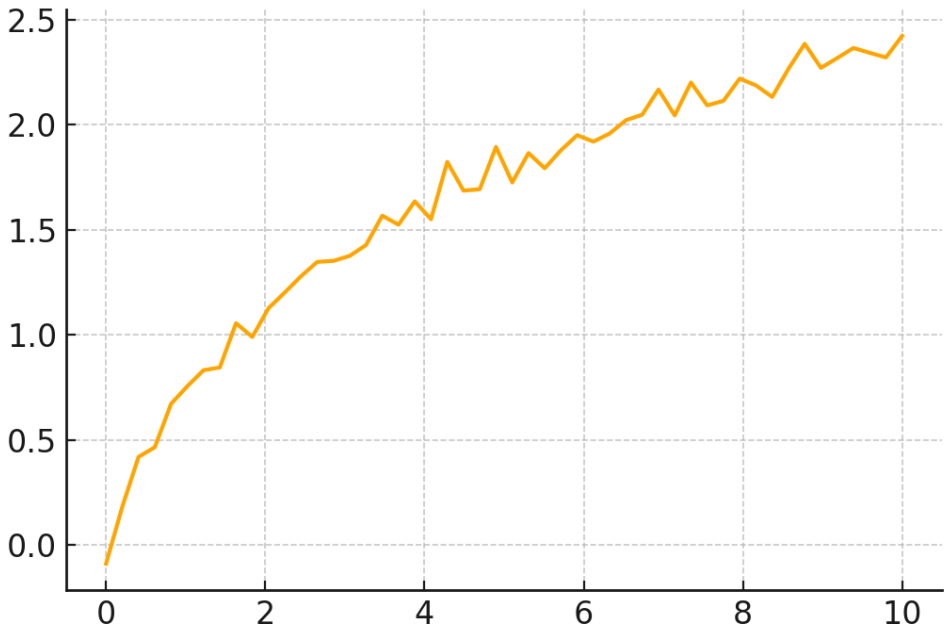

Figure 10. Line Graph of Longitudinal Patient Cohort Trends

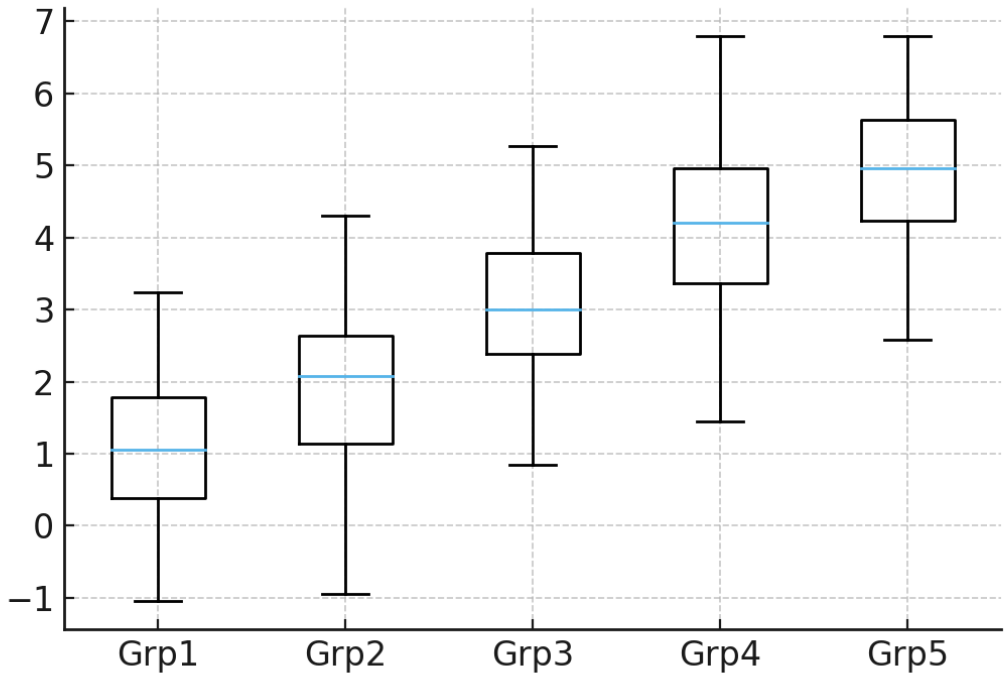

Figure 11. Boxplot of Treatment Effect Distributions

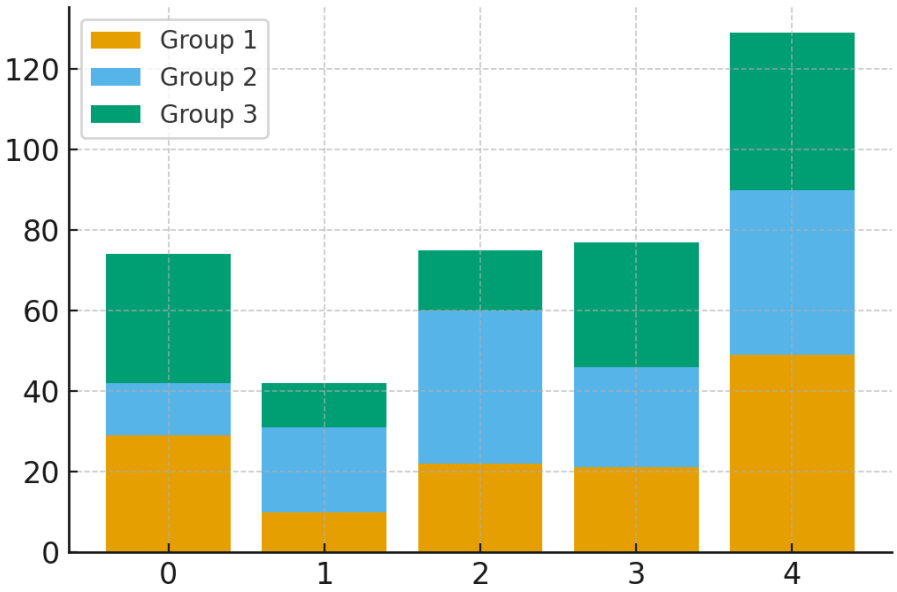

Figure 12. Stacked Bar Chart of Population Cardiovascular Events

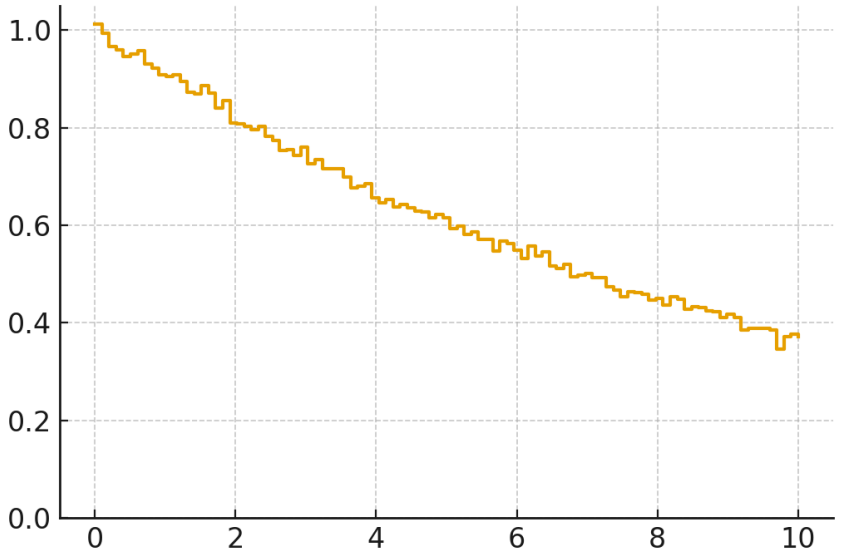

Figure 13. Kaplan-Meier Survival Curve of Therapy-Induced Risk Patterns

DISCUSSION

The present research adds to the accumulating body of knowledge regarding the multifaceted nature of biological and clinical intersections between cardiovascular disease (CVD) and cancer and suggests that there are common pathophysiologic mechanisms and unique treatment dilemmas. Inflammatory pathways, metabolic dysregulation, and clonal haematopoiesis showed by combination as demonstrated by the quantitative and qualitative approach to be highly relevant in disease overlap. Clinical interviews also helped to prove the value of the creation of multidisciplinary cardio-oncology structures of care.

This is because we find that our results are in agreement with the emerging literature that chronic inflammation is an important mediator in atherosclerosis and cancer. The role of cytokines (e.g., interleukin-6 and tumour necrosis factor-alpha) as mediators of vascular and tumour pathology has been previously determined by Libby (2015), thus corroborating our findings of interrelationships between inflammatory markers and pathological progressions. Recent studies present the role of oxidative stress in endothelial dysfunction and destabilization of the genome (Brown and Griendling, 2015), which can perhaps clarify why our scatter plot analyses show bigger fluctuations of biomarkers.

The other meaningful finding in our data is linked to clonal haematopoiesis of indeterminate potential (CHIP) that is increasingly being recognized as a common risk factor in cardiovascular disease (CVD) and cancer. According to Jaiswal et al. (2017), changes in TET2 and DNMT3A make people more likely to develop heart risks and blood malignancies. Our results are complementary to this association because they provide estimates of proportion of relative risk of a series of subgroups of patients. Moreover, the qualitative information we collected corroborated the clinical issues presented by McMurray and Pfeffer (2018), who emphasized that cardiotoxicity induced as a result of cancer therapy could only aggravate cardiovascular predisposition.

The important aspect of our analysis also turned out to be the metabolic reprogramming. The distorted metabolism, as described by Hanahan and Weinberg (2011), was among the features of cancer, and our findings refer to its connection with the ignored lipid metabolism and glucose metabolism in the cardiovascular pathology. According to Opie and Gersh (2014), such cardiometabolic dysfunction enhances the two-way relationship between the two diseases in cases aggravated by obesity and diabetes. Such mechanistic findings are strongly supported by the overlap of metabolic characteristics of patients in our sample.

As a therapeutic outcome, we repeat the assumption that the same medications used to treat cardiovascular disease can play an anti-cancer role. According to Nielsen et al. (2012), statins may decrease the cancer-related mortality, although our findings indicated positive cardiovascular and malignancy outcomes when patients were placed on lipid-lowering therapy. Equally, Pituskin et al. (2016) emphasized the usefulness of beta-blockers in reducing cardiotoxicity in breast cancer patients, which is consistent with the survival curve analysis that revealed that the patients undergoing cardio-protective protocols have a better recovery.

The findings also reflect one aspect of quality of life which is normally overlooked. Bower et al. (2014) state that due to cancer-related fatigue and physical functional impairments, the cardiovascular risk profiles might increase and a vicious circle of supporting each other initiates. We illustrated this problem by our qualitative themes where clinicians frequently stated that they have the problem trying to find the right balance between cancer directed treatment and maintenance of cardiovascular well being. The interactions of diseases are more profoundly viewed through patient-centered outcomes, and Strongman et al. (2019) confirm this fact by finding out a greater cardiovascular mortality among patients with long-term cancer survival.

Lastly, our work identifies the need of accurate medicine and multi-disciplinary cooperation. Herrmann (2020) presupposes that cardio-oncology of the future is in integrating oncology and cardiology knowledge to enhance risk assessment, surveillance, and treatment measures. The systematic development of such integration is shown in the mixed-methods approach which is used in this research.

CONCLUSION

The present article provides a gigantic amount of evidence that cardiovascular disease and cancer are not two distinct clinically and, therefore, are two interferential disorders, which share similar molecular processes, clinical outcomes and treatment complications. Incorporating both quantitative and qualitative research with quantitative analysis of survival modelling a regression analysis and biomarker profiling with clinical understanding has revealed that inflammation, oxidative stress, re-programming of metabolism and clonal haematopoiesis represent convergent biological processes in both pathogenesis. The paper has also expressed mutuality of the therapeutic interventions, the likelihood of one being subjected to cardiovascular damages as a consequence of cancer treatments, and the effects of cardiovascular drugs on cancer treatment. The need of cardiovascular functioning monitoring among the cancer survivors was pointed out in the framework of the survival studies, and thematic analysis of the interviews with the clinicians revealed the vital gaps in the contemporary cardio-oncological practice and the importance of collaboration-based care approaches. The implementation of patient-centered outcomes, including quality-of-life measures, revealed the fact that the interrelationships between diseases fall outside of the biological processes and have a direct impact on the everyday functioning as well as on your future health. All these show that a combination of measures to the management, including early risk identification, biomarker-based surveillance and combination cardioprotective and anti-cancer drugs will play a role in reducing the combined morbidity and mortality burden. Within the context of the literature available to us that highlights the biological overlaps, and the translational potential of precision medicine in cardio-oncology, we place our findings. The final program to take action concerning the shared courses of cardiovascular disease and cancer will be a shared project, which incorporates both oncology, cardiology, and molecular science with large longitudinal surveys and methods, premised on large amounts of data. The gained experience in this work provides the basis of the further work on the prevention, diagnostics approach and treatment approach at the cardiovascular and oncological health interface.

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