

Original Article

A Randomized Trial of miRNA-34a-Based Therapy for Chemosensitization in Triple-Negative Breast Cancer Patients

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ABSTRACT

Background: Triple-negative breast cancer is an aggressive malignancy with limited targeted treatment options and variable response to neoadjuvant chemotherapy. Restoring tumor-suppressive miRNA-34a activity may improve chemosensitivity. **Objective:** To determine whether adding a liposomal miRNA-34a mimic to standard neoadjuvant chemotherapy increases pathological complete response in stage II–III triple-negative breast cancer. **Methods:** This prospective, two-arm randomized controlled trial was conducted at a tertiary cancer Centre in urban Sindh, Pakistan, from May 2025 to January 2026. Eighty-four women were randomized equally to dose-dense AC-T chemotherapy with weekly intravenous liposomal miRNA-34a mimic or chemotherapy alone. Pathologists were blinded to allocation. Outcomes were analyzed for 80 participants with postoperative data using chi-square tests, t-tests, repeated-measures analysis of variance, and Pearson correlation. **Results:** Pathological complete response occurred in 62.5% of intervention participants and 35.0% of controls (risk difference, 27.5%; 95% CI, 6.4–48.6; $p=0.014$). Residual tumor diameter was lower with combination therapy (1.1 ± 0.8 vs 2.2 ± 1.1 cm; mean difference, -1.10 cm; 95% CI, -1.53 to -0.67 ; $p<0.001$). The time-by-group interaction for tumor regression was significant ($F(2,156)=14.86$; $p<0.001$). **Conclusion:** Liposomal miRNA-34a combined with neoadjuvant chemotherapy demonstrated a preliminary improvement in pathological and imaging responses, although confirmation requires complete randomized and safety analyses. **Keywords:** Chemosensitization; MicroRNA-34a; Neoadjuvant Therapy; Pathological Complete Response; Triple-Negative Breast Cancer.

INTRODUCTION

Triple-negative breast cancer (TNBC) is an aggressive breast cancer subtype characterized by the absence of estrogen receptor and progesterone receptor expression and human epidermal growth factor receptor 2

overexpression. It accounts for approximately 15%–20% of breast cancer diagnoses and is associated with limited molecularly targeted treatment options, early recurrence, and an increased risk of distant metastasis. Because endocrine and HER2-directed therapies are ineffective in this population, systemic chemotherapy remains a principal component of treatment, particularly for patients with stage II–III disease receiving neoadjuvant therapy. Pathological complete response (pCR), defined by the absence of residual invasive cancer in the breast and regional lymph nodes after neoadjuvant treatment, provides an important measure of therapeutic response in TNBC (1).

Anthracycline- and taxane-based chemotherapy can reduce tumor burden and facilitate surgical management; however, treatment response varies considerably among patients. A substantial proportion of patients have residual disease after neoadjuvant chemotherapy because of intrinsic or acquired resistance involving altered apoptotic signaling, tumor-cell plasticity, survival pathways, and changes in non-coding RNA regulation. These mechanisms can enable malignant cells to tolerate cytotoxic injury and contribute to incomplete pathological response and subsequent disease progression. MicroRNAs have consequently received increasing attention as regulators of breast cancer biology, treatment sensitivity, and resistance to systemic therapy (2,3).

MicroRNAs are short, non-coding RNA molecules that regulate gene expression at the post-transcriptional level and can influence several interconnected cellular pathways. Within these regulatory networks, miRNA-34a has been recognized as a tumor-suppressive microRNA associated with the control of cellular proliferation, apoptosis, survival, and treatment response. Reduced or dysregulated miRNA-34a expression may contribute to the persistence of resistant tumor-cell populations, whereas restoration of its activity has been proposed as a strategy for increasing susceptibility to anticancer treatment. Unlike interventions directed at a single protein, miRNA-based approaches may affect multiple signaling pathways simultaneously, providing a biological rationale for investigating miRNA-34a replacement as an adjunct to chemotherapy (1,3–5).

Despite this therapeutic rationale, the clinical translation of miRNA mimics remains challenging. Systemically administered RNA molecules are susceptible to degradation, inefficient tumor delivery, rapid clearance, off-target effects, and treatment-related toxicity. Liposomal delivery systems have been developed to improve the stability and cellular uptake of therapeutic miRNAs, but the clinical evidence supporting their efficacy remains limited. Existing literature has primarily described the molecular functions, biomarker potential, delivery challenges, and preclinical activity of miRNA-based therapies rather than their comparative clinical effectiveness when combined with standard neoadjuvant chemotherapy (4,5). Therefore, it remains uncertain whether systemic restoration of miRNA-34a can meaningfully increase pathological tumor clearance in patients with TNBC.

To address this knowledge gap, the present prospective, parallel-group randomized controlled trial compared standard dose-dense doxorubicin–cyclophosphamide followed by paclitaxel chemotherapy combined with a weekly intravenous liposomal miRNA-34a mimic against the same chemotherapy regimen alone in women with untreated stage II–III TNBC. The primary objective was to determine whether adding the miRNA-34a mimic increased the postoperative pCR rate, defined as ypT0/is ypN0. The secondary objectives were to compare changes in primary tumor diameter and serum miRNA-34a-related molecular markers between the treatment groups. It was hypothesized that the combination regimen would produce a higher pCR rate and greater tumor regression than standard neoadjuvant chemotherapy alone.

MATERIALS AND METHODS

A prospective, two-arm, parallel-group randomized controlled trial was conducted to determine whether adding a liposomal miRNA-34a mimic to standard neoadjuvant chemotherapy improved treatment response in women with triple-negative breast cancer. The randomized design was selected to compare the therapeutic effect of the combination regimen with standard chemotherapy while reducing selection bias and balancing prognostic characteristics between the groups. The study was conducted at a specialized tertiary cancer centre in urban Sindh, Pakistan, from May 2025 through January 2026. The study period covered participant screening, randomization, a 16-week treatment phase, surgical resection and postoperative histopathological assessment.

Women aged 25–65 years with histologically confirmed, previously untreated stage II or III triple-negative breast cancer were assessed for eligibility. Participants were required to have an Eastern Cooperative Oncology Group performance status of 0 or 1 and adequate organ function for systemic chemotherapy. Patients were excluded if they had previously received systemic chemotherapy, had an active autoimmune disorder or severe hepatic

impairment, or had pre-existing peripheral neuropathy exceeding grade 1. Potentially eligible patients presenting to the study centre during the recruitment period were screened against these criteria. Of the 102 patients screened, 84 met the eligibility requirements, agreed to participate and were enrolled. The target sample of 84 participants was based on the anticipated difference in treatment response reported in previous translational RNA-therapy research, with equal allocation planned between the treatment groups.

A computer-generated randomization sequence using variable block sizes was used to allocate participants in a 1:1 ratio. Allocation concealment was maintained using sequentially numbered, opaque, sealed envelopes held by an independent trial pharmacist. Forty-two participants were assigned to receive standard dose-dense doxorubicin-cyclophosphamide followed by paclitaxel chemotherapy combined with a liposomal miRNA-34a mimic, and 42 were assigned to receive the same chemotherapy regimen alone. Participants remained in their originally allocated groups when treatment adherence, discontinuation and outcome availability were documented.

Participants in both groups received four cycles of intravenous doxorubicin and cyclophosphamide administered every two weeks, followed by four cycles of intravenous paclitaxel administered every two weeks. The complete dose-dense AC-T regimen was delivered over 16 weeks. Participants allocated to the intervention group additionally received a weekly intravenous infusion of the liposomal miRNA-34a mimic at 1.5 mg/kg throughout the same 16-week treatment period. The control group received no miRNA-34a mimic. Double blinding was not feasible because of the preparation and administration requirements of the investigational formulation. Treating personnel were therefore aware of treatment allocation, whereas the surgical oncologists responsible for postoperative assessment and the pathologists evaluating resection specimens were blinded to group assignment.

Participants were monitored weekly throughout treatment to evaluate treatment adherence, protocol discontinuation and adverse events. Adverse events were classified according to the Common Terminology Criteria for Adverse Events, version 5.0. Tumor response was assessed by magnetic resonance imaging at baseline, week 8 and week 16. Tumor size was recorded as the maximum primary tumor diameter in centimetres. Surgical resection was undertaken following completion of neoadjuvant treatment, and postoperative tissue assessment was performed at approximately week 18. Resected breast and axillary lymph-node specimens were evaluated microscopically by blinded pathologists.

The primary outcome was pathological complete response after neoadjuvant treatment, defined as the absence of residual invasive carcinoma in both the resected breast tissue and axillary lymph nodes, corresponding to ypT0/is ypN0. Secondary outcomes comprised the change in primary tumor diameter from baseline to week 16, the trajectory of tumor regression across baseline, week 8 and week 16, serum miRNA-34a expression and BCL-2 suppression. Serum specimens were evaluated using quantitative real-time polymerase chain reaction. Serum miRNA-34a expression was summarized as fold change, while BCL-2 suppression was expressed as a percentage. The association between post-treatment serum miRNA-34a expression and residual tumor diameter was also examined in the intervention group.

Random sequence generation, concealed allocation and blinded pathological and surgical outcome assessment were used to reduce selection and ascertainment bias. Both groups received the same dose-dense AC-T regimen over the same treatment period, with the addition of the miRNA-34a mimic representing the planned difference between groups. Weekly monitoring was used to evaluate treatment adherence and identify adverse events. Standardized outcome timing and maintenance of original treatment-group labels were used to support consistency in data collection and analysis.

Continuous variables were assessed for normality using the Shapiro-Wilk test and summarized as mean and standard deviation when the distributional assumption was satisfied. Categorical variables were summarized as frequencies and percentages. Independent-samples t-tests were used to compare continuous variables between groups, and chi-square tests were used to compare categorical variables, including the pathological complete response rate. Within-group changes in tumor diameter from baseline to week 16 were evaluated using paired-samples t-tests. Repeated-measures analysis of variance was used to examine the effects of time, treatment group and the time-by-group interaction on tumor diameter across baseline, week 8 and week 16. Pearson's correlation coefficient was used to evaluate the relationship between post-treatment serum miRNA-34a expression and residual tumor diameter in the intervention group. All statistical tests were two-tailed, and $p < 0.05$ was considered statistically significant.

Baseline characteristics were summarized for all 84 randomized participants. Postoperative efficacy analyses were based on the 80 participants with available outcome measurements, comprising 40 participants in each group. Four randomized participants without postoperative assessments—two from the intervention group and two from the control group—were excluded from the available-case efficacy analysis. No replacement values were used for these missing postoperative observations.

RESULTS

During the total study duration of eight months (May 2025 to January 2026), 102 patients with stage II–III triple-negative breast cancer were screened for eligibility at the tertiary care facility in Urban Region Sindh. Of these, 18 individuals were excluded (12 did not meet inclusion criteria, and 6 declined participation). The remaining 84 patients were enrolled and randomly assigned in a 1:1 ratio to receive either standard neoadjuvant chemotherapy combined with liposomal miRNA-34a mimic (intervention group, N=42) or standard neoadjuvant chemotherapy alone (control group, N=42).

The active intervention phase lasted 16 weeks across both groups, followed by surgical resection and histopathological review. Prior to surgery, 4 participants were lost to follow-up or discontinued the protocol (2 in the intervention group due to treatment-unrelated relocation, and 2 in the control group due to toxicity-induced treatment cessation). Thus, a final analyzed sample of 80 participants (intervention, n=40; control, n=40) completed the study and were evaluated on an intention-to-treat basis for post-operative outcomes.

Baseline demographic and clinical parameters were recorded for the full randomized sample (N=84). As summarized in Table 1, no statistically significant differences were observed between the intervention and control groups regarding mean age (51.4 ± 7.2 vs. 50.8 ± 6.9 years, $p=0.697$), baseline primary tumor diameter (4.8 ± 1.1 vs. 4.6 ± 1.0 cm", $p=0.388$), ECOG performance status distribution ($p=0.825$), or clinical tumor stage ($p=0.811$), establishing complete baseline comparability across groups.

Histopathological evaluation of surgical specimens from the final analyzed sample (n=80) revealed a significantly higher primary outcome rate of pathological complete response (ypT0/is ypN0) in the intervention group compared to the control group (62.5%, n=25/40 vs. 35.0%, n=14/40; absolute risk difference: 27.5%, 95% CI" : 6.4% to 48.6%, $p=0.014$). Post-intervention tumor diameter measurements showed a significant between-group mean difference favoring the intervention group (1.1 ± 0.8 vs. 2.2 ± 1.1 cm" ; mean difference: -1.1 cm", 95% CI" : -1.53 to -0.67 cm" , $p<0.001$), as detailed in Table 2. Within-group paired t-tests confirmed significant primary tumor volume reductions from baseline to week 16 in both the intervention group (mean reduction: -3.7 ± 1.0 cm" , $p<0.001$) and control group (mean reduction: -2.4 ± 0.9 cm" , $p<0.001$), presented in Table 3.

Table 1: Baseline Demographic and Clinical Characteristics of Participants (N = 84)

Parameter	Total Sample (N=84)	Intervention Group (N=42)	Control Group (N=42)	p-value
Age (years), Mean $\pm$ SD	51.1 ± 7.0	51.4 ± 7.2	50.8 ± 6.9	0.697
Tumor Diameter (cm), Mean $\pm$ SD	4.7 ± 1.1	4.8 ± 1.1	4.6 ± 1.0	0.388
ECOG Performance Status, n (%)				0.825
Grade 0	52 (61.9%)	27 (64.3%)	25 (59.5%)	0.811
Grade 1	32 (38.1%)	15 (35.7%)	17 (40.5%)	
Clinical Stage, n (%)				
Stage II	58 (69.0%)	28 (66.7%)	30 (71.4%)	0.811
Stage III	26 (31.0%)	14 (33.3%)	12 (28.6%)	

Table 2: Post-Intervention Comparison of Primary Outcomes (n=80)

Outcome Measure	Intervention Group (n=40)	Control Group (n=40)	Mean Difference / Risk Diff. (95% CI)	p-value
Pathological Complete Response (pCR), n (%)	25 (62.5%)	14 (35.0%)	27.5% (6.4% to 48.6%)	0.014
Residual Tumor Diameter (cm), Mean $\pm$ SD	1.1 ± 0.8	2.2 ± 1.1	-1.10 (-1.53 to -0.67)	<0.001

Table 3: Within-Group Pre-Post Changes in Tumor Diameter (n=80)

Group	Baseline (cm), Mean $\pm$ SD	Week 16 (cm), Mean $\pm$ SD	Mean Change (cm)	Paired t-test (p-value)
Intervention Group (n=40)	4.8 ± 1.1	1.1 ± 0.8	-3.7 ± 1.0	<0.001
Control Group (n=40)	4.6 ± 1.0	2.2 ± 1.1	-2.4 ± 0.9	<0.001

Table 4: Secondary Outcomes (Target Marker Expression at Week 16, n=80)

Marker Outcome	Intervention Group (n=40)	Control Group (n=40)	Between-Group Difference	p-value
Serum miRNA-34a (Fold-Change), Mean ± SD	3.45±0.82	1.05±0.21	2.40 (2.13" to " 2.67)	<0.001
BCL-2 Suppression Rate (%), Mean ± SD	58.4±12.1	21.2±8.6	37.2 (32.5" to " 41.9)	

Repeated measures ANOVA conducted across baseline, week 8, and week 16 demonstrated significant time main effects ($F(2,156)=214.35$, $p<0.001$), group main effects ($F(1,78)=18.42$, $p<0.001$), and a significant time-by-group interaction effect for primary tumor volume regression ($F(2,156)=14.86$, $p<0.001$), confirming a faster rate of tumor regression in the intervention arm. Additionally, serum expression markers evaluated in Table 4 demonstrated a markedly greater increase in circulating miRNA-34a target levels post-intervention in the miRNA-34a group (3.45 ± 0.82 fold-change) relative to controls (1.05 ± 0.21 fold-change; $p<0.001$). Pearson correlation analysis revealed a strong inverse relationship between post-treatment serum miRNA-34a levels and residual tumor diameter in the intervention arm ($r=-0.68$, $p<0.001$).

DISCUSSION

The addition of a liposomal miRNA-34a mimic to standard neoadjuvant chemotherapy was associated with a higher pathological complete response rate, smaller residual tumor diameter, a more pronounced tumor-regression trajectory, increased circulating miRNA-34a expression, and greater BCL-2 suppression. These findings address the study objective by suggesting that miRNA-34a replacement may enhance the short-term response to anthracycline- and taxane-based chemotherapy in patients with stage II–III triple-negative breast cancer. Nevertheless, the efficacy findings were based on the 80 participants with available postoperative data rather than the complete randomized population of 84 participants.

The observed treatment pattern is biologically consistent with previous literature identifying dysregulated microRNA expression as an important component of TNBC progression, prognosis, and chemotherapy resistance. Earlier evidence indicates that microRNAs regulate interconnected pathways governing apoptosis, proliferation, tumor-cell survival, invasion, and response to systemic therapy. Reduced tumor-suppressive microRNA activity may therefore permit malignant cells to tolerate cytotoxic injury, while restoration of selected microRNAs could potentially increase treatment susceptibility (1–3). However, the available references predominantly comprise mechanistic, preclinical, and narrative evidence rather than directly comparable randomized clinical trials. The present findings should consequently be interpreted as preliminary clinical evidence rather than confirmation of an established therapeutic effect.

A plausible explanation for the observed findings is that restoration of miRNA-34a activity altered the balance between pro-survival and pro-apoptotic signaling during chemotherapy. MicroRNAs can regulate multiple messenger RNA targets simultaneously, potentially limiting the ability of malignant cells to maintain compensatory survival pathways after cytotoxic exposure (2,3,5). The greater BCL-2 suppression observed in the intervention group is compatible with reduced anti-apoptotic signaling, while the inverse correlation between serum miRNA-34a expression and residual tumor diameter supports a relationship between molecular exposure and tumor response. Nevertheless, the correlation does not demonstrate that circulating miRNA-34a directly reached tumor tissue, caused BCL-2 suppression, or mediated the pathological response. Confirmation of this mechanism would require validated tumor-tissue measurements, longitudinal molecular sampling, and a formal mediation analysis.

The liposomal formulation may have contributed to the observed molecular response by protecting the miRNA mimic from degradation and facilitating cellular delivery. Existing research has identified stability, tissue delivery, rapid clearance, off-target activity, and systemic toxicity as major barriers to the clinical development of miRNA-based anticancer treatments (4,5). Although the present study recorded efficacy and molecular signals in favour of the combination regimen, it did not provide a complete arm-specific analysis of adverse events, serious adverse events, dose modifications, or treatment interruptions. Two control-group participants discontinued treatment because of toxicity, but the nature, grade, attribution, and outcome of these events were not reported. The benefit–risk profile of the investigational regimen therefore cannot be determined from the available results.

From a clinical perspective, an improvement in pathological complete response could be meaningful in a disease characterized by limited treatment options and frequent residual disease after neoadjuvant chemotherapy. The absolute difference in pCR observed in the available-case analysis suggests a potentially important treatment

signal. However, pCR remains an intermediate pathological endpoint, and the study did not evaluate recurrence, event-free survival, overall survival, quality of life, or late toxicity. The results therefore do not establish that miRNA-34a treatment improves long-term prognosis or should be incorporated into routine clinical practice. The findings instead support further evaluation of the therapeutic concept under appropriately registered, regulated, and adequately monitored clinical-trial conditions.

The principal strengths of the study were its randomized parallel-group design, equal allocation, use of the same dose-dense AC-T regimen in both groups, allocation concealment, blinded pathological assessment, and inclusion of pathological, imaging, and molecular outcomes. The pCR definition was clinically recognizable, and repeated tumor assessment permitted evaluation of response over the treatment period. The observed concordance between pathological response, residual tumor diameter, circulating miRNA-34a expression, and BCL-2 suppression also provides internally supportive evidence across different outcome domains.

Several limitations substantially affect interpretation. The study was conducted at one centre with a modest sample, limiting precision and generalizability. Four randomized participants were excluded from the postoperative efficacy analysis, meaning that the reported analysis was available-case rather than intention-to-treat. Although attrition was numerically balanced, its differing causes and the absence of sensitivity analyses leave uncertainty regarding the effect of missing outcomes. Participants and treating clinicians were not blinded, creating the possibility of performance bias. The investigational formulation, standard chemotherapy doses, molecular-assay procedures, sample-size assumptions, protocol registration, and regulatory oversight were incompletely described. Furthermore, week-8 descriptive tumor measurements were not presented, despite their inclusion in the repeated-measures analysis. The secondary outcomes and correlation analysis were not accompanied by a stated multiplicity strategy, and the intervention-group correlation may have been exploratory. Finally, incomplete adverse-event reporting and the short observation period prevent conclusions regarding safety and long-term clinical benefit.

Overall, the results provide a preliminary efficacy signal supporting continued investigation of miRNA-34a replacement as a potential chemosensitizing strategy in TNBC. Confirmation will require a prospectively registered multicentre trial with a reproducible investigational-product description, analysis of all randomized participants, prespecified missing-data and multiplicity procedures, complete adverse-event reporting, validated molecular assays, and longer follow-up. Such evidence is necessary to determine whether the observed pathological and imaging responses translate into a favourable benefit-risk profile and improved patient outcomes.

CONCLUSION

Among participants with available postoperative outcome data, adding a weekly liposomal miRNA-34a mimic to dose-dense AC-T neoadjuvant chemotherapy was associated with a higher pathological complete response rate, smaller residual tumor diameter, and more pronounced molecular changes than chemotherapy alone. These findings indicate a potentially important chemosensitizing effect, but the incomplete randomized analysis, limited sample, absent comprehensive safety results, and lack of long-term outcomes prevent conclusions regarding routine clinical effectiveness, safety, or prognosis.

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