Prognosis and Management of DCIS with Microinvasion: Insights from SEER Database and a Chinese Cohort

Xiuxing Chen[1†], Qing Peng[1†], Ying Zhong[2†], Zhuo Li[1], Guoying Liu[1], Herui Yao[1*], Yaping Yang[1*]
[1]Sun Yat-Sen Memorial Hospital, Sun Yat-Sen University ; [2] Hui Ya Hospital of The First Affiliated Hospital, Sun Yat-Sen University

SAN ANTONIO BREAST CANCER SYMPOSIUM® DECEMBER 9-12, 2025 HENRY B. GONZALEZ CONVENTION CENTER • SAN ANTONIO, TX
UT Health San Antonio Mays Cancer Center AACR American Association for Cancer Research®
ID : PS4-08-02

Background

- Ductal carcinoma in situ with microinvasion (DCISM) represents approximately 1% of all breast cancers and 5–10% of DCIS cases. It poses a diagnostic and therapeutic challenge, bridging non-invasive and invasive carcinoma.
- Existing evidence lacks consensus on its prognosis or optimal management.
- Our study leveraged large-scale SEER and Chinese CSCO datasets to clarify DCISM characteristics, outcomes, and treatment efficacy.

Methods and Materials

- This retrospective cohort study analyzed data from two sources: the Surveillance, Epidemiology, and End Results (SEER) database (1998–2018), comprising 7,467 patients (4,458 with DCIS and 3,009 with DCISM), and a Chinese cohort of 1,132 patients (903 with DCIS and 229 with DCISM) from Sun Yat-sen Memorial Hospital and the Chinese Society of Clinical Oncology. (**Figure 1**)
- To reduce confounding bias, risk regression analysis and propensity score matching were applied, enabling a robust comparison of survival outcomes between DCIS and DCISM.
- Key variables included patient age, tumor size, molecular subtypes (ER/PR status), and treatment modalities (surgical type, radiotherapy, chemotherapy, and endocrine therapy). Prognostic endpoints assessed were overall survival (OS), breast cancer-specific survival (BCSS), and disease-free survival (DFS).

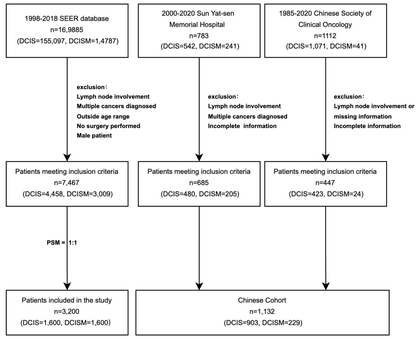


Figure 1. Flowchart of Patient Selection.

Results

Clinicopathological Characteristics of Patients

- Patients with DCISM displayed more aggressive pathological characteristics than those with DCIS, including a higher prevalence of high-grade tumors (P < 0.001) and elevated HER2 expression (P < 0.001), alongside a greater likelihood of undergoing total mastectomy (P < 0.001).
- Despite these differences, no statistically significant disparities were observed in OS, BCSS, or DFS between the two groups, suggesting comparable long-term prognoses. (**Figure 2**)

Adverse prognostic factors

- Older age (DCIS: HR = 4.35, P = 0.013; DCISM: HR = 5.07, P = 0.025) and BCS alone (DCIS: HR = 2.37, P = 0.003; DCISM: HR = 3.84, P = 0.0002) were linked to worse OS, while radiotherapy improved survival in both DCIS and DCISM. Age and treatment remained independent prognostic factors. **(Table 1)**

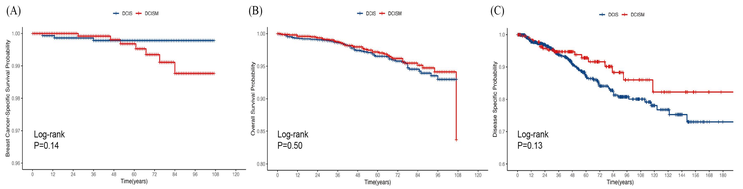


Figure 2. Comparative survival outcomes between DCIS and DCISM.
(A) All Patients; (B) DCIS; (C) DCISM

(A) All patients

Variable	Hazard ratio	P value
Univariate Cox regression analysis		
Age (>=50 vs. <50)	4.59 (1.87–11.29)	0.0009
Race		
African American vs. White	1.31 (0.77–2.22)	0.3134
African vs. White	0.51 (0.21–1.25)	0.1417
Others vs. White	NA	0.9942
Pathological grade (Nicbporrly vs. Well/moderately)	0.95 (0.62–1.45)	0.8037
T stage (DCISM vs. DCIS)	0.87 (0.59–1.30)	0.5019
Primary tumor size (>=2cm vs. <2cm)	NA	0.9946
ER (Positive vs. Negative)	0.81 (0.51–1.29)	0.3768
PR (Positive vs. Negative)	1.00 (0.65–1.53)	0.9849
HER2 (Positive vs. Negative)	0.70 (0.44–1.13)	0.1416
Breast subtype		
HER2+/HR+ vs. HER2−/HR+	0.57 (0.30–1.09)	0.0895
HER2+/HR− vs. HER2−/HR+	1.01 (0.53–1.91)	0.9648
HER2−/HR− vs. HER2−/HR+	1.37 (0.74–2.54)	0.3177
Surgery (non-BCS vs. BCS)	1.09 (0.67–1.78)	0.7302
Radiotherapy (Yes vs. No)	0.42 (0.28–0.63)	<0.0001
Chemotherapy (Yes vs. No)	0.49 (0.12–1.98)	0.3151
Therapy		
BCS vs. BCS+Radiotherapy	2.90 (1.86–4.51)	<0.0001
RM Therapy vs. BCS+Radiotherapy	1.65 (0.96–2.84)	0.0701
Multivariate Cox regression analysis		
Age (>=50 vs. <50)	4.49 (1.82–11.04)	0.0011
Therapy		
BCS vs. BCS+Radiotherapy	2.83 (1.82–4.41)	<0.0001
RM Therapy vs. BCS+Radiotherapy	1.71 (1.00–2.95)	0.0512

(B) DCIS

Variable	Hazard ratio	P value
Univariate Cox regression analysis		
Age (>=50 vs. <50)	4.35 (1.36–13.91)	0.0133
Race		
African American vs. White	1.08 (0.53–2.21)	0.8301
African vs. White	0.17 (0.02–1.20)	0.0778
Others vs. White	NA	0.9951
Pathological grade (Nicbporrly vs. Well/moderately)	0.89 (0.50–1.57)	0.6799
Primary tumor size (>=2cm vs. <2cm)	NA	0.9950
ER (Positive vs. Negative)	0.79 (0.43–1.48)	0.4672
PR (Positive vs. Negative)	1.02 (0.56–1.81)	0.9411
HER2 (Positive vs. Negative)	0.82 (0.45–1.50)	0.5165
Breast subtype		
HER2+/HR+ vs. HER2−/HR+	0.67 (0.30–1.51)	0.3340
HER2+/HR− vs. HER2−/HR+	1.14 (0.51–2.57)	0.7468
HER2−/HR− vs. HER2−/HR+	1.29 (0.54–3.06)	0.5694
Surgery (non-BCS vs. BCS)	1.04 (0.47–2.30)	0.9185
Radiotherapy (Yes vs. No)	0.47 (0.28–0.80)	0.0055
Chemotherapy (Yes vs. No)	NA	0.9957
Therapy		
BCS vs. BCS+Radiotherapy	2.37 (1.36–4.16)	0.0025
RM Therapy vs. BCS+Radiotherapy	1.96 (0.92–4.18)	0.0802
Multivariate Cox regression analysis		
Age (>=50 vs. <50)	4.37 (1.33–13.66)	0.0144
Therapy		
BCS vs. BCS+Radiotherapy	2.34 (1.34–4.10)	0.0030
RM Therapy vs. BCS+Radiotherapy	1.48 (0.64–3.44)	0.3590

(C) DCISM

Variable	Hazard ratio	P value
Univariate Cox regression analysis		
Age (>=50 vs. <50)	5.07 (1.23–20.99)	0.0250
Race		
African American vs. White	1.67 (0.77–3.64)	0.1969
African vs. White	1.02 (0.36–2.88)	0.9768
Others vs. White	NA	0.9970
Pathological grade (Nicbporrly vs. Well/moderately)	1.01 (0.54–1.92)	0.9668
Primary tumor size (>=2cm vs. <2cm)	NA	0.9963
ER (Positive vs. Negative)	0.83 (0.41–1.69)	0.6077
PR (Positive vs. Negative)	0.97 (0.51–1.86)	0.9282
HER2 (Positive vs. Negative)	0.56 (0.26–1.20)	0.1340
Breast subtype		
HER2+/HR+ vs. HER2−/HR+	0.46 (0.16–1.31)	0.1441
HER2+/HR− vs. HER2−/HR+	0.82 (0.29–2.33)	0.7070
HER2−/HR− vs. HER2−/HR+	1.48 (0.62–3.58)	0.3791
Surgerynon-BCS vs. BCS)	1.19 (0.62–2.28)	0.6030
Radiotherapy (Yes vs. No)	0.36 (0.19–0.68)	0.0016
Chemotherapy (Yes vs. No)	0.68 (0.16–2.81)	0.5890
Therapy		
BCS vs. BCS+Radiotherapy	3.84 (1.87–7.89)	0.0002
RM Therapy vs. BCS+Radiotherapy	1.96 (0.92–4.18)	0.0802
Multivariate Cox regression analysis		
Age (>=50 vs. <50)	4.88 (1.18–20.25)	0.0289
Therapy		
BCS vs. BCS+Radiotherapy	3.68 (1.79–7.58)	0.0004
RM Therapy vs. BCS+Radiotherapy	2.09 (0.96–4.45)	0.0562

Table 1. Prognostic factors associated with overall survival in DCIS and DCISM.
(A) All Patients; (B) DCIS; (C) DCISM

Results

Treatment of DCISM and DCIS

- **Surgery and radiation**: BCS combined with radiotherapy emerged as the optimal treatment strategy for both DCISM and DCIS, significantly enhancing DFS compared to BCS alone or radical mastectomy (DCIS: BCS alone vs. BCS + radiotherapy, HR = 2.37, P = 0.003; DCISM: HR = 15.03, P = 0.015). In DCISM, this approach also improved OS (HR = 3.85, P < 0.001). (**Figure 3**)

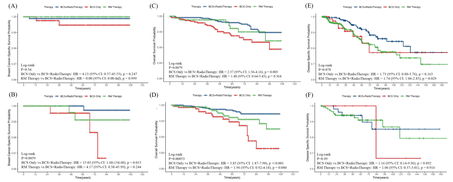


Figure 3. Comparison of survival outcomes according to surgical and radiotherapeutic strategies.
(A) DCIS (SEER): BCSS; (B) DCISM (SEER): BCSS; (C) DCIS (SEER): OS; (D) DCISM (SEER): OS; (E) DCIS (CSCO): DFS; (F) DCISM (CSCO): DFS

- **Chemotherapy**: Post-surgical chemotherapy was associated with significantly worse DFS in DCISM patients (P = 0.0062), indicating a potential detrimental effect, whereas it showed no significant impact on BCSS or OS in DCIS patients. (**Figure 4**)

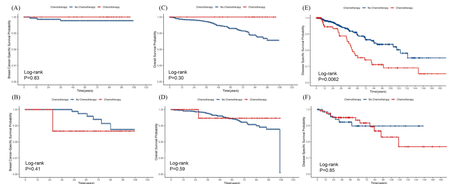


Figure 4. Impact of chemotherapy on survival outcomes.
(A) DCIS (SEER): BCSS; (B) DCISM (SEER): BCSS; (C) DCIS (SEER): OS; (D) DCISM (SEER): OS;(E) DCIS (CSCO): DFS; (F) DCISM (CSCO): DFS

- Endocrine therapy markedly improved DFS in DCIS patients (HR = 0.46, P = 0.013), but this benefit was not evident in DCISM.
- Targeted therapy yielded no clear survival advantage in HER2-positive cases (P > 0.05).

Conclusions

- DCISM shows similar survival to DCIS despite more aggressive pathology.
- Patients benefit more when accepted BCS plus radiation regardless with or without microinvasion.
- Chemotherapy was not recommended for DCISM patients.

Contact

Yaping Yang, MD, PhD,
Guangdong Provincial Key Laboratory of Malignant Tumor Epigenetics and Gene Regulation, Breast Tumor Center, Sun Yat-sen Memorial Hospital, Sun Yat-sen University, No. 107 Yanjiang West Road, Guangzhou 510120, China (E-mail: yangyap2@mail.sysu.edu.cn);
And
Herui Yao, MD, PhD,
Guangdong Provincial Key Laboratory of Malignant Tumor Epigenetics and Gene Regulation, Breast Tumor Center, Sun Yat-sen Memorial Hospital, Sun Yat-sen University, No. 107 Yanjiang West Road, Guangzhou 510120, China (E-mail: yaoherui@mail.sysu.edu.cn).