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What can we learn from the age- and race/ethnicity- specific rates of inflammatory breast carcinoma?

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Abstract

Inflammatory Breast Carcinoma (IBC), the most aggressive type of breast tumor with unique clinicopathological presentation, is hypothesized to have distinct etiology with a socioeconomic status (SES) component. Using the Surveillance, Epidemiology and End Results (SEER) Program data for 2004–2007, we compare incidence rates of IBC to non-inflammatory locally advanced breast cancer (LABC) among racial/ethnic groups with different SES. The analysis includes women 20–84 years of age. To examine evidence for the distinct etiology of IBC, we analyzed age-distribution patterns of IBC and non-inflammatory LABC, using a mathematical carcinogenesis model. Based on the Collaborative Staging Extension codes, 2,942 incident IBC cases (codes 71 and 73) and 5,721 non-inflammatory LABC cases (codes 40–62) were identified during the four-year study period. Age-adjusted rates of IBC among non-Hispanic White and Hispanic women were similar (2.5/100,000 in both groups). Similar rates were also found in non-inflammatory LABC in these two groups (4.8/100,000 and 4.2/100,000, respectively). In African-American women, the IBC (3.91/100,000) and non-inflammatory LABC (8.47/100,000) rates were greater compared with other ethnic/racial sub-groups. However, the ratio of rates of IBC/non-inflammatory LABC was similar among all the racial/ethnic groups, suggesting that African-American women are susceptible to aggressive breast tumors in general but not specifically to IBC. The mathematical model successfully predicted the observed age-specific rates of both examined breast tumors and revealed distinct patterns. IBC rates increased until age 65 and then slightly decreased, whereas non-inflammatory LABC rates steadily increased throughout the entire age interval. The number of critical transition carcinogenesis stages (m-stages) predicted by the

model were 6.3 and 8.5 for IBC and non-inflammatory LABC, respectively, supporting different etiologies of these breast tumors.

Keywords

Epidemiology; Inflammatory breast cancer; Etiology

Introduction

Inflammatory breast cancer (IBC) is a rare subtype of the locally advanced breast cancer (LABC) with distinct clinicopathological characteristics [1]. It is considered to be the most aggressive form of breast cancer with rapid progression and poor survival [1]. In the United States, the proportion of incident IBC ranges from 1 to 5% of all breast cancer cases. In 2008 the First International Conference on Inflammatory Breast Cancer emphasized to recognize IBC as a distinct entity with the goal to develop guidelines for the management of IBC [2, 3]. As such, IBC is likely to have a different etiology from the non-inflammatory LABC [4].

With largely unknown etiology, IBC remains to be a mysterious, aggressive disease. Previous studies include case-only analyses [1, 5–8], and analyses of data from the Surveillance, Epidemiology and End Results (SEER) Program and the North American Association of Central Cancer Registries (NAACCR) [9–13]. The Tunisian and SEER data show that higher proportions of IBC arise among younger women [5, 10, 13]. In addition, the Tunisian data show that a higher proportion of IBC cases arises in the rural regions [likely those of low socioeconomic status (SES)]. Interestingly, the occurrence of IBC plummeted with the increase in the gross domestic product (GDP) during the last several decades [5]. Based on these divergent trends in Tunisia and the high prevalence of rural cases, it has been hypothesized that IBC may have an environmental component related to SES. In the United States, IBC rates have been greater among African-Americans for the period up to 2000 [10, 13, 14] (a racial group with lower SES [15]).

In summary, the previously published analyses shaped a consensus that the age at diagnosis of IBC is younger compared with the non-inflammatory breast cancer and that there is a racial disparity in IBC rates, suggesting that this disparity may stem from the SES inequality [15]. However, there are several facts that are not consistent with this consensus. For example, aggressive breast tumors are associated with lower SES, as shown in California (1999–2004), where both African-American and Hispanic women were more likely to have aggressive breast tumors as well as to live in socioeconomically deprived areas [16]. In contrast, IBC rates according to the SEER data (1994–1998) were lower among Hispanic than among African-American women and were similar to those found in non-Hispanic white women [9]. This finding does not support the hypothesized relation of SES and IBC, because African-Americans and Hispanics have similar rates of poverty, which are approximately threefold greater than Whites [17].

It is possible that a specific SES component responsible for greater IBC rates among African-Americans has a different prevalence among the Hispanic women, whereas the SES components responsible for the greater rates of other subtypes of aggressive breast tumors have similar prevalence among both minority populations. If so, then the IBC rates should differ between African-American and Hispanic women, but the rates of non-inflammatory LABC—also an aggressive type of breast tumor—should be similar in these two groups. To examine this assumption, we assessed the race/ethnicity-specific IBC and noninflammatory

LABC rates using the most recent available SEER data for 2004–2007 [18]. The limitation of the data to later than 2004 is based on the change of IBC coding [19].

To address the question of distinct etiology of IBC from non-inflammatory LABC, we used population-based biologically motivated modeling to examine the patterns of age-specific incidence rates for these two aggressive subtypes of breast tumors.

Methods

Data Source

We used population-based data from the National Cancer Institute's SEER program that covers approximately 26% of the population in the United States [20]. Specifically, we used SEER 17 registry databases and Hurricane Katrina Impacted Louisiana cases (November 2009) that were released in April 2010 [18].

Definition of IBC Case and Selection of Comparison Group

IBC cases were defined as primary breast carcinoma (C50.0–50.9) using the Collaborative Staging (CS) Extension codes 71 and 73 according to the American Joint Committee on Cancer (AJCC) sixth edition [21]. This definition is based on the changes in the coding of IBC since 2004 [19]. Accordingly, the definition of noninflammatory LABC included breast cancer cases with the CS Extension codes 40–62. The definition for the CS Extension codes used in this study is the following:

“IBC—71 = Diagnosis of inflammatory carcinoma WITHOUT a clinical description of inflammation, erythema, edema, peau d'orange, etc., of more than 50% of the breast, with or without dermal lymphatic infiltration, inflammatory carcinoma, NOS; 73 = Diagnosis of inflammatory carcinoma with a clinical description of inflammation, erythema, edema, peau d'orange, etc., of more than 50% of the breast, WITH or WITHOUT dermal lymphatic infiltration;

Non-inflammatory LABC—40 = Invasion of (or fixation to): Chest wall; Intercostal or serratus anterior muscle (s), rib (s); 51 = Extensive skin involvement, including: Satellite nodule(s) in skin of primary breast; Ulceration of skin of breast; Any of the following conditions described as involving not more than 50% of the breast, or amount or percent of involvement not stated; Edema of skin; En cuirasse; Erythema; Inflammation of skin; peau d'orange (“pigskin”), 52 = Any of the following conditions described as involving more than 50% of the breast WITHOUT a stated diagnosis of inflammatory carcinoma: Edema of skin; En cuirasse; Erythema; Inflammation of skin; peau d'orange (“pigskin”); 61 = (40) + (51); 62 = (40) + (52)”.

Because most previous reports used the definition of IBC based on the TNM staging as T4d, we examined the agreement between the two IBC definitions. For IBC with T4d definition, the comparison group of non-inflammatory LABC was all cases with codes T4 and T4a–c. Our study population was restricted to women diagnosed between 2004 and 2007 with tumors of known malignant behavior and a known age at diagnosis.

Study Variables

The current analysis concentrated on age-adjusted incidence rates stratified by race and ethnicity and on age-specific incidence rates for each age from 20 to 84. Women 85 years old or older were not included in the analysis, because those with the age at diagnosis of 85 and older were lumped into one category in the SEER data and therefore, age-specific rates could not be calculated for this group. Two variables were used to define race and ethnicity:

race (White, Black, and other) and origin (Hispanic and Non-Hispanic). Patients with missing race and/or origin characteristics or characteristics that were missing or coded as “other or unknown” were not included in the analysis.

Statistical Analysis

The SEER 17 Registries database was received from the National Cancer Institute in both ASCII and SEER*Stat format. Age-specific rates were generated using SEER*stat software and cross checked by the calculation using ASCII format by the SAS statistical software package (SAS for Windows Version 9.2, SAS Institute Inc., Cary, NC). Age-adjusted race/ethnicity-specific incidence rates and their 95% confidence intervals (CIs) were estimated using the SEER*Stat software package version 6.6.2. Incidence rates were expressed per 100,000 woman-years and were age-adjusted by the direct method to the 2000 United States population. The hypothesis that race/ethnic subgroups are more susceptible to IBC was tested by comparing the ratios of IBC to non-inflammatory LABC rates between the subgroup; the standard error (SE) for the IBC/nonI-LBC rate ratio was estimated as SE for the ratio of two uncorrelated variables:

$$\sigma_r = \frac{r_{IBC}}{r_{LABC}} \left(\frac{\sigma_{IBC}^2}{r_{IBC}^2} + \frac{\sigma_{LABC}^2}{r_{LABC}^2} \right)^{1/2}, \quad (1)$$

where r —represents rates and σ^2 —represents variance.

The carcinogenesis model used for the analysis of age-specific rates combines the concept of random frailty with the base Armitage-Doll model. The key assumption of the frailty component is heterogeneity of human populations with respect to the rate of carcinogenic mutations, representing individual predisposition to cancer that is randomly distributed. Application of the frailty model to cancer risk used the Armitage-Doll base model that operates under the assumption that cancer results from accumulation of a critical number of mutations (m -stages). Manton et al. [22] and Kravchenko et al. [23] applied this combined model to the analysis of SEER data focusing on evaluating Armitage-Doll m -stages for different cancers based on age-dependent patterns of specific types of cancer. The explicit equation of the model is for incidence (I) dependence on age (x):

$$I_0(x) = \frac{x^{m-1}}{c^m (1 + n\sigma^2 c^{-m} m^{-1} x^m)^{1/n}}, \quad (2)$$

where the four parameters are: (a) the number of carcinogenesis stages (m), (b) the scale parameter inversely related to the rates of the transitions between m -stages (c , years), (c) the variance of frailty distribution (σ^2) reflecting individual susceptibility to cancer risk, and (d) the parameter (n) describing the shape of the frailty distribution ($n = 1, 2$, and 0 corresponds to gamma-distribution, inverse Gaussian distribution, and the distribution suggested by Manton et al. [24], respectively). For $n = 1$, the shape of the age-pattern represented by the model has a maximum with age equal to

$$c \left(m(m-1)(n+m-mn)^{-1} \sigma^{-2} \right)^{1/m} \quad (3)$$

This model was used to examine the patterns of age-specific rates comparing IBC and non-inflammatory LABC and to calculate m -stages for these breast cancer subtypes.

Results

Both definitions of IBC, i.e., T4d and CS Extension code 71 and 73, identified 2,942 incident IBC cases diagnosed among women 20–84 years of age during the period of 2004–2007. The numbers of non-inflammatory LABC cases identified by the two definitions were slightly different: 5,755 incident non-inflammatory LABC cases were identified based on CS Extension codes 40–62 and 5,721 cases were identified based on T4 and T4a–c TNM staging. With such a small difference, we used a more inclusive definition for non-inflammatory LABC cases based on the CS extension code. Thus, our analysis included 2,942 incident IBC cases and 5,755 incident non-ILABC cases.

Cross-tabulation between race and origin variables revealed that almost all IBC cases (99.7%) can be classified as Non-Hispanic White, Non-Hispanic Black, Hispanic White, or others. Hispanic Black women presented only 0.3% of IBC cases (Table 1). In further analysis we used the above named race/ethnicity categories. The highest rates of both cancer types were greater among African-American women and similar among White women of Hispanic and non-Hispanic origin (Table 2). The relative rates of IBC among all racial/ethnic groups were similar, suggesting that specific susceptibility to IBC as compared with non-inflammatory LABC does not vary between these groups (Table 2).

Modeling age-specific rates of IBC and non-inflammatory LABC revealed two distinct patterns. The rates of IBC increased up to age 60, peaked around age 65, and then slightly declined (Fig. 1). The existence of this peak was predicted by the parameter n from the IBC model (Eq. 2): $n = 0.95$, which is below one and therefore, predicting that there is a maximum for age-specific rates. Furthermore, the age of maximum IBC rate that was calculated using Eq. 3 was 65.7 years, which corresponded well to the observed data. The non-inflammatory LABC rates continued to increase throughout the entire examined age range (Fig. 1). For this subtype, the model predicted no peak with $n = 1.61$. The numbers of m -stages derived from the models were significantly different: the calculated number of stages was 6.3 (95% CI, 5.8–6.8) and 8.5 (95% CI, 7.3–9.8) for IBC and non-inflammatory LABC, respectively (Fig. 1). The random distribution of the residuals from both models indicated that the models performed reasonably well. The ratio of the rates (IBC/non-inflammatory LABC) changed dramatically throughout the age range from above one until 30 years of age and declining to approximately 0.25 at older ages. Such change in the relative rates indicates that the specific susceptibility to IBC precipitates in younger ages (Fig. 2).

Discussion

In our analysis, the new standards of IBC coding based on the CS Extension codes corresponded well to the TNM criteria (T4d) used in the previous analyses of the SEER data as both definitions of IBC produced exactly the same number of cases. The rates of IBC are comparable with what was found in a previously published study by Hance et al. [10], whereas the rates for non-inflammatory LABC observed in this analysis were greater. Several differences in the analysis explain this inconsistency. The definition of non-inflammatory LABC in our analysis includes T4 along with T4a–c, whereas the previous SEER analysis [10] includes only T4a–c. Our analysis was restricted to ages 20–84, whereas Hance et al. [10] did not use any age restriction. Finally, our analysis did not include any tumor characteristics, whereas Hance et al. analyzed tumor characteristics and restricted the data to only those cases with known tumor characteristics.

Our analysis (Table 2) confirms earlier findings of greater rates of IBC found in African-American as compared with White women [9, 10], and similar rates among Hispanic and

Non-Hispanic White women [9]. Close rates observed for non-inflammatory LABC among Hispanic and Non-Hispanic Whites contradicts the earlier findings from California which indicated that African-American and Hispanic women were more likely to have aggressive breast tumors and to live in socioeconomically deprived areas [16]; however, this contradiction can be explained by the specific SES structure of this particular geographic region. Similar rate ratios of IBC/non-inflammatory LABC indicate that the susceptibility to aggressive tumors in general, but not specifically to IBC, is associated with race. We envision two (not mutually exclusive) explanations for these findings: (i) prevalence of a specific SES component associated with IBC is similar among Hispanic and non-Hispanic Whites, but greater among African-Americans; or (ii) some genetic component predisposes African-American women to both aggressive tumor types. To address the question as to whether genetic or SES components or a combination of both drive the racial differences in IBC rates, an analytic epidemiological study should be conducted.

The analysis of age-specific rates shows a clear difference in the age-specific patterns of IBC and non-inflammatory LABC rates (Fig. 1). The deceleration of IBC rates around age 65 suggests that the prevalence of some etiological component for IBC declines around menopausal age (Fig. 1). This assumption is supported by the earlier analysis of age density histograms of IBC and noninflammatory LABC cases, showing that the major inflection points for IBC and non-inflammatory LABC are at age 50 and 74, respectively [10]. Note that there is approximately a 15-year interval between the inflection point determined by the age density analysis by Hance et al. [10] and decline in age-specific rates of IBC in our analysis (Fig. 1). According to the expectation that the decline in age-specific rates occurs later, we did not detect a decline of age-specific rates in non-inflammatory LABC until age 84 (Fig. 1). The existence of an age-dependent etiological component for IBC is also suggested by the dynamics of the rate ratio of IBC to non-inflammatory LABC, specifically by its rapid decline until approximately age 35 and less rapid decline after 35 (Fig. 2). We speculate that these findings reflect specific susceptibility of women during pregnancy and lactation to IBC. This assumption is supported by the findings in Tunisia that IBC incidence precipitates around first pregnancy and lactation [5]. The age-period 20–35 is associated with first pregnancy and lactation, suggesting that changes in hormonal milieu contribute to IBC etiology. This is not surprising as exposure to estrogen was established as the main risk factor for breast cancer more than a century ago [25]. However, the connection of estrogen and to IBC risk versus non-inflammatory breast cancer (including noninflammatory LABC) probably involves different etiological mechanisms. The main evidence pointing to such difference is an inverse association between non-inflammatory breast cancer and early pregnancy, whereas the existing case-only analysis suggests a positive association of early pregnancy with IBC. To fully understand whether estrogen-related risk factors for IBC differ from noninflammatory LABC, a case-control or a cohort study had to be conducted.

The additional evidence for a distinct etiology of IBC comes from the different number of m -stages predicted by the biologically motivated mathematical models for IBC and non-inflammatory LABC: 6.3 vs 8.5. The exact biological meaning of m -stages is not specified by the model; such stages may constitute a critical number of events that lead to cancer onset, such as somatic mutations, epigenetic changes, or changes in the microenvironment. A lower number of m -stages predicted by the IBC model corresponds to the observed age-specific rate pattern with higher rates of IBC at ages before 30 and to the clinical observation of extremely rapid onset of the disease [1]. The differences in the molecular phenotype of IBC and noninflammatory LABC support our findings which indicate different etiological pathways for these breast cancer subtypes. Compared with non-IBC mammary tumors, IBC tumors have higher frequency of HER2 overexpression cancer, increased cytoplasmic MUC1 staining, increased expression of *E*-cadherin and rhoC, and most

importantly a mutation called LIBC (Lost in IBC), which encodes a low affinity insulin-like growth factor 1 (IGF1) binding protein (IGFBP-9) [1].

In summary, our findings serve as a platform to generate an etiological hypothesis for IBC. The higher IBC rates among African-American women are not specific to IBC, but may rather reflect predisposition to aggressive breast tumors. Whether this predisposition entails specific SES and/or cultural and/or genetic components is an important question that can be best answered by a case-control population-based study. Based on the risk factors known for breast cancer in general, younger age of diagnosis suggests a genetic component, but there are no data addressing this question. Finally, we would like to note that despite the distinct susceptibility of women at younger ages to IBC, the majority of IBC cases occur after age 55 (>50%), suggesting that a different constellation of risk factors may be responsible for the variation in the age of diagnosis. It is logical to assume that the risk factors specific to IBC may cluster in younger ages, whereas IBC etiology at later ages may also include risk factors common to non-inflammatory LABC. The specific risk factors for IBC and the role of the classical risk factors for breast cancer in this disease cannot be assessed from SEER data or from the case-only comparison. Because IBC is such an aggressive disease and is distinctly different from other types of breast cancer, it needs careful examination in a classical epidemiological population-based case-control study.

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Abbreviations

CI	Confidence interval
CS extension	Collaborative staging extension
GDP	Gross domestic product
IBC	Inflammatory breast cancer
LABC	Locally advanced breast cancer
NAACCR	North American Association of Central Cancer Registries
SEER Program	Surveillance, Epidemiology and End Results Program
SES	Socioeconomic status

Fig. 1. Age-specific rates of IBC (*left panel*) and non-inflammatory LABC (*right panel*) obtained from SEER data for 2004–2007. To show in more detail the shape of the age-specific pattern of IBC rates, a window with age-specific IBC rates on a different scale is included in the *left panel*. Line shows prediction by the model, *open circles* present observed age-specific rates. *Closed circles* present residuals

Fig. 2. Ratio of IBC age-specific rates to non-inflammatory LABC rates obtained from SEER data for 2004–2007. *Line* shows prediction by the model, *closed circles* show the ratio of age-specific rates during a five-year interval

Table 1
Race/ethnicity distribution of IBC and non-inflammatory LABC incident cases identified by the SEER in 2004–2007

Race	Ethnicity	IBC number (%)	Non-inflammatory LABC number (%)
Whites	Non-Hispanic	1920 (65%)	3729 (65%)
	Hispanic	375 (13%)	580 (10%)
Blacks	Non-Hispanic	444 (15%)	927 (16%)
	Hispanic	9 (0.3%)	7 (0.1%)
Other		194 (6.6%)	512 (8.9%)
Total		2942 (100%)	5755 (100%)

Non-Hispanic non-Spanish-Hispanic-Latino, *Hispanic* Spanish-Hispanic-Latino

Table 2
Rates of IBC and non-inflammatory LABC by race/ethnicity

	IBC		Non-inflammatory LABC		Population	Rate ratio IBC/non-inflammatory LABC (95% CI)
	Count	Rate (95% CI)	Count	Rate (95% CI)		
All	2,942	2.59 (2.50–2.69)	5755	5.06 (4.92–5.19)	110,051,306	0.51 (0.46–0.56)
Blacks	453	3.91 (3.56–4.30)	934	8.47 (7.93–9.05)	12,240,993	0.46 (0.35–0.57)
Non-Hispanic Whites	1,920	2.54 (2.42–2.65)	3729	4.77 (4.62–4.93)	66,536,935	0.53 (0.47–0.59)
Hispanic Whites	375	2.51 (2.26–2.80)	580	4.17 (3.83–5.45)	18,667,066	0.60 (0.35–0.87)
Other	194	1.59 (1.37–1.83)	512	4.27 (3.91–4.66)	12,606,312	0.37 (0.24–0.50)

Incidence rates are expressed as number of incident cases per 100,000 adjusted for age