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Case report

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Structural dedifferentiation of breast cancer arising within the major lactiferous ducts: imaging-histopathologic correlation in two fatal cases, part IV

Abstract

Background: Breast cancers originating in the major lactiferous ducts provide considerable detection and treatment challenges. These malignancies comprise a disproportionately high fraction of all breast cancer deaths. We have studied the pathophysiology of these cancers and described three distinct phases in their evolution. The 1st phase begins within the pre-existing, major duct as an *in situ* process that is occult on imaging. The malignancy progresses to the 2nd phase when new, branching, duct-like structures (neoducts, termed ductal adenocarcinoma of the breast, DAB) develop and proliferate. These neoducts have the specific hallmarks of duct forming invasive carcinoma, including central necrosis with amorphous calcifications and architectural distortion, both of which facilitate their detection at imaging. The final, 3rd phase of progression involves further structural dedifferentiation, in which cancer cells escape from the cancer-filled neoducts through breaches in the boundaries of the neoducts (dedifferentiated DAB, ddDAB). The extraductal cancer cells and fragments of the neoducts are both able to spread through the interstitial space and metastasize to distant organs.

Precis: These detailed case reports contain correlative illustrations of comprehensive breast imaging and large format histopathology depicting structural dedifferentiation of breast cancers originating in the major lactiferous ducts. These examples, all with long-term follow-up, have been chosen to help the reader understand the pathophysiology of this complex, often-fatal process.

Case description: Case 1 presents a 52-year-old woman with a self-detected, firm superficial tumour in the upper-outer quadrant of her right breast, which reminded her of a "frozen pea", small and hard at palpation. Multimodality imaging showed multiple invasive tumour foci. Preoperative 14G core biopsy established the diagnosis of invasive dedifferentiated DAB. Sliced specimen radiographs correlated with large format thin-section histopathology images revealed multiple 2nd phase invasive cancer-filled neoducts (DAB) and adjacent 3rd phase tumour foci (invasive dedifferentiated DAB), documenting the evolution of cancer developing within the major ducts. The patient underwent a partial mastectomy followed by chemotherapy, radiotherapy and endocrine therapy. Forty-five months after diagnosis and initiation of treatment, the patient was diagnosed with liver metastases. The histopathologic and immunohistochemical (IHC) biomarkers of the liver metastases closely resembled those of the previously diagnosed breast cancer. Five years and five months after diagnosis, the patient died from disseminated dedifferentiated DAB.

Case 2 describes a 66-year-old woman who had previously undergone bilateral breast reduction surgery with scar tissue causing extensive bilateral architectural distortion and post-traumatic fat necrosis-type calcifications detectable on her postoperative screening mammograms. These findings resulting from surgical trauma posed a considerable diagnostic challenge for the breast imagers. Retrospective application of an AI algorithm gave inconsistent results for this series of screening examinations. A decade after her surgery and twenty-five months after her most recent screening mammograms she presented with a self-detected firm breast tumour in the upper-outer quadrant of her right breast. Following 14G core biopsy the image-guided histopathologic workup established the diagnosis of DAB and ddDAB. The large format histopathologic images of the mastectomy specimen show the close proximity of cancer-filled neoducts (DAB) to the multiple invasive foci of the escaped dedifferentiated DAB cells. The patient was treated with mastectomy, chemotherapy and Herceptin without radiotherapy or endocrine therapy. Twenty-eight months after diagnosis and initiation of treatment the patient was diagnosed with bronchial metastases. Four years and four months after diagnosis and treatment the patient died from metastasizing neoducts (DAB) and invasive, dedifferentiated DAB.

Conclusion: These and our previously published cases illustrate the three phases of evolution of cancers originating within the major lactiferous ducts and emphasize that the 2nd phase, conventionally called ductal carcinoma *in situ*, DCIS, is a potentially harmful, duct forming invasive carcinoma. It is the precursor of the 3rd phase and is not *in situ*. Because the 2nd phase is classified by histopathology terminology as DCIS, HER2 testing is not part of routine work-up under current guidelines, so HER2-positive cases in this phase remain unidentified in clinical practice. Consequently, the current terminology constrains research to evaluate the use of Herceptin treatment at an earlier phase of this aggressive disease subgroup, when it could prevent or delay progression through structural dedifferentiation to the 3rd phase. The combination of two invasive processes, the 2nd and 3rd phases, is particularly harmful and often fatal.

Keywords: mammography, breast cancer neoducts, neoductgenesis, dedifferentiation of neoducts, imaging biomarkers

Volume 17 Issue 4 - 2026

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Received: October 30, 2026 | **Published:** November 12, 2026

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List of abbreviations: DCIS, ductal carcinoma *in situ*; DAB, ductal adenocarcinoma of the breast; ddDAB, dedifferentiated DAB; H&E, haematoxylin-eosin staining; IHC, immunohistochemical; MLO, medio-lateral oblique projection; CC, cranio-caudal projection; ER, estrogen receptor; PR, progesterone receptor; HER2, human epidermal growth factor receptor 2; Ki-67, antigen Kiel 67; EGFR, epidermal growth factor receptor; GATA3, GATA binding protein 3, CK, cytokeratin staining; TTF-1, thyroid transcription factor 1; TDLU, terminal ductal-lobular unit

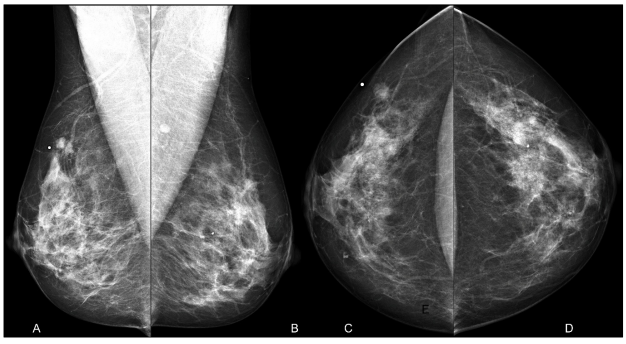
Introduction

The review of fatal breast cancers can show us our current limitations in diagnosis and therapy and should lead to the following questions: 1) What are the specific causes of death in these patients? 2) What can we do to help prevent similar breast cancer deaths? In the two cases presented here, the fatal metastases appeared several years after the patient had received surgical and systemic therapy. The deaths of women given care according to standard guidelines should lead us to review the guidelines and re-evaluate the therapy given in these specific cases. In our series of 309 women with fragmented casting type calcifications on the mammogram, 55 died from breast cancer. The breast cancer-specific survival of these women was significantly poorer than that of women with grade-matched tumours of apparent acinar origin (HR 2.54, 95% CI 1.09-6.04), and this poor survival did not improve between 1996-2007 and 2008-2022.[1]

We have approached this problem by analysing our breast cancer cases according to the apparent site of origin of each breast tumour, determined by correlating the imaging biomarkers with large section histopathology images. By following the vital status of all our breast cancer patients for decades, we have been able to publish reports on the significantly higher breast cancer mortality of patients with cancers arising from the major lactiferous ducts.[1-6] Our refined assessment of the cause of death determines the characteristic histopathologic and immunohistochemical features of each fatal primary breast tumour, allowing comparison with the corresponding features in the distant metastases.[7]

First case

A 52-year-old woman presented with an approximately 10 mm firm, palpable tumour mass located in the upper-outer quadrant of her right breast. Mammographic workup and hand-held ultrasound showed multifocal invasive tumour foci. Preoperative 14G core biopsy established the diagnosis of invasive dedifferentiated DAB (Figure 1). The patient underwent a partial mastectomy followed by chemotherapy, radiotherapy and endocrine therapy. Specimen radiographs correlated with large format histopathology images revealed multifocal collections of dedifferentiated DAB (Figure 2). The close proximity of DAB (the cancer-filled neoducts) with dedifferentiated DAB (ddDAB) is striking and indicative of their successive phases of development (Figure 3).



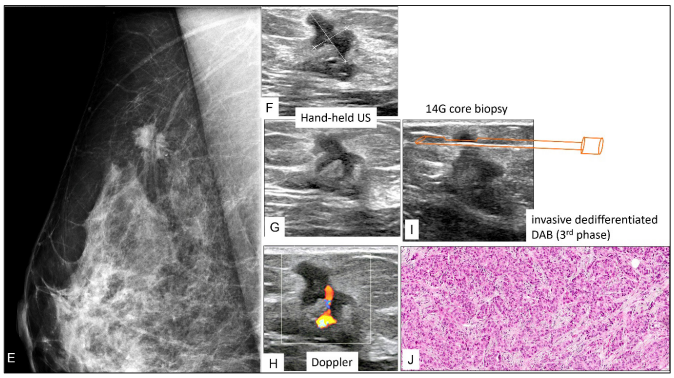
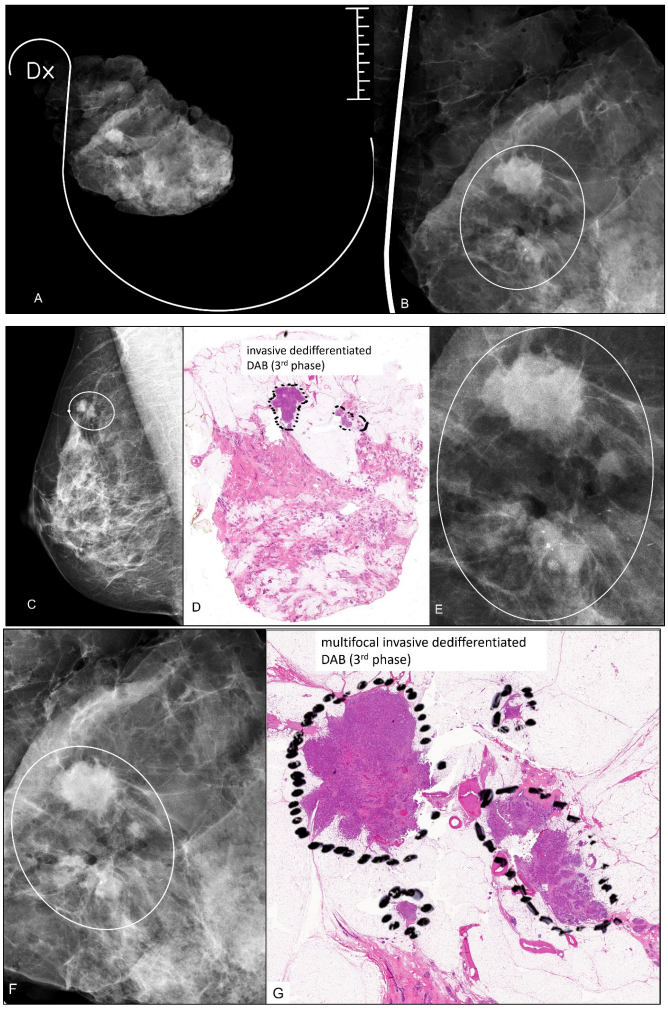


Figure 1 Medio-lateral (A,B) and craniocaudal (C,D) mammograms. Seven cm from the nipple, in the upper-outer quadrant of the right breast, the lead pellet points to the small, palpable, circular tumour. Microfocus magnification mammography (E) and hand-held ultrasound images (F-H) confirm the presence of multifocal tumours. 14G core biopsy (I). Microscopic examination of the core specimen shows extraductal invasive tumour foci (dedifferentiated DAB) (J).



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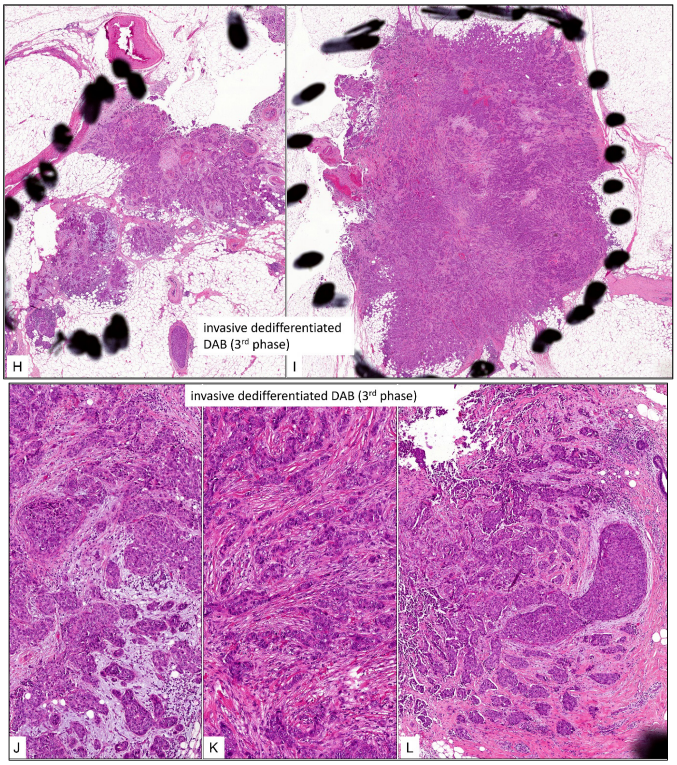
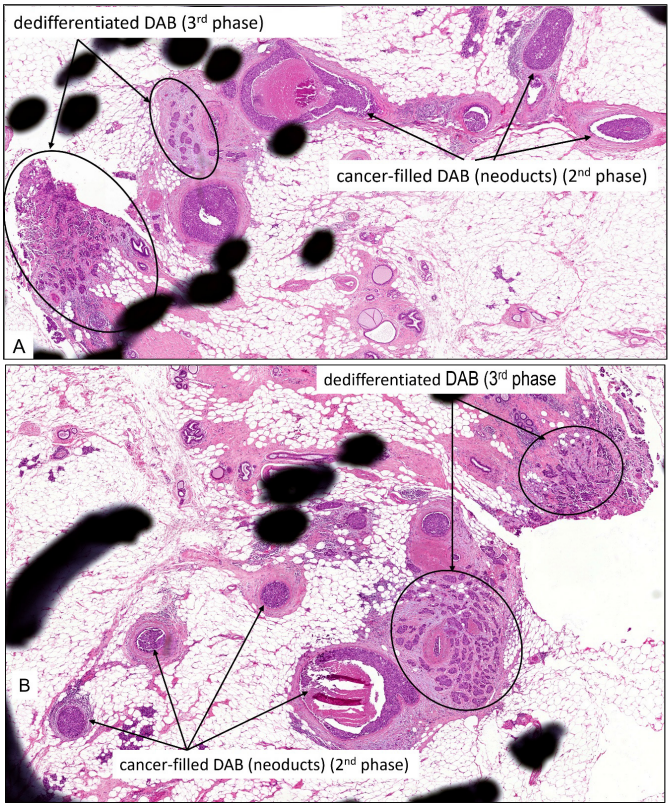


Figure 2 Specimen radiograph without (A) and with microfocus magnification (B) demonstrating multifocality. The right MLO projection (C) and the microfocus magnification specimen radiographs (E,F) are correlated with the large format histopathology images (D,G). Intermediate-and higher power histopathology images (H-L) illustrate the details of the collections of ddDAB cells having escaped from the neoducts (3rd phase in the progression of cancers originating within the major ducts).



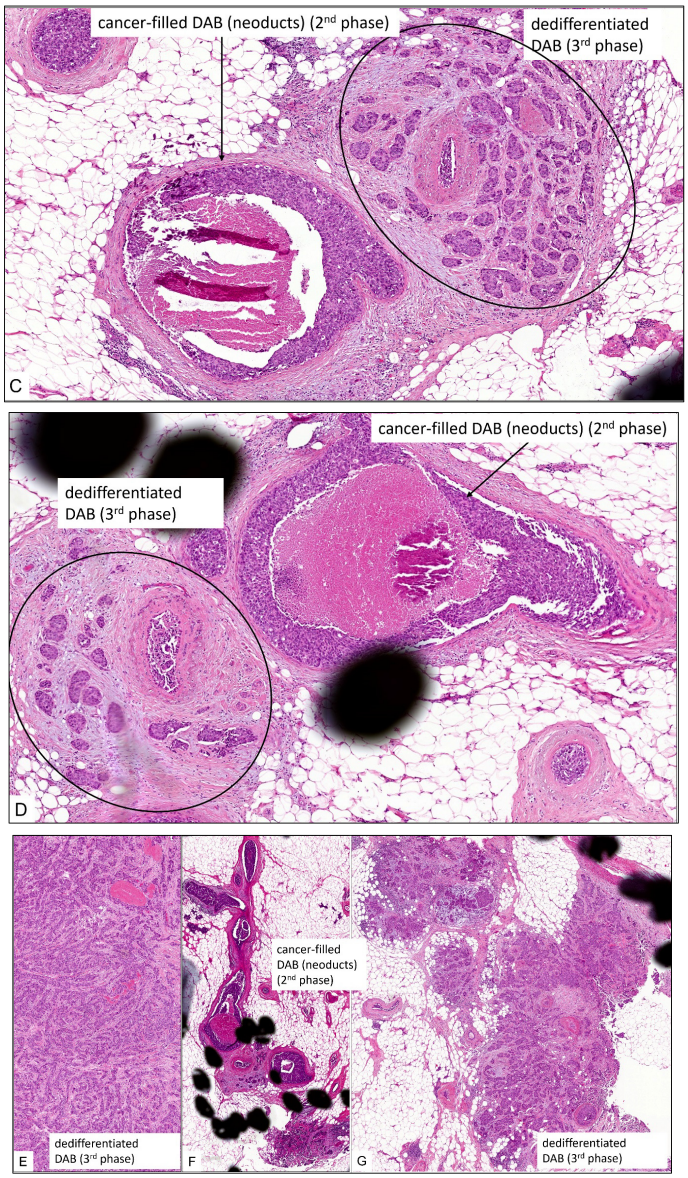


Figure 3 A series of large format thin section histopathology images (A-G) illustrating the close proximity of the cancer-filled neoducts (2nd phase) with the invasive dedifferentiated DAB (3rd phase) of the evolution of cancer developing within the major ducts.

The official histopathology diagnosis: Multifocal, poorly differentiated invasive ductal carcinoma. The individual foci measured 12x10 mm, 11x10 mm and additional smaller satellites in the size range of 2 and 1 mm, associated with Grade 3 DCIS. Total disease extent: 30x20 mm. IHC biomarkers: ER positive, PR negative, HER2 negative, Ki-67 24%, E-cadherin positive, CK 5/6, CK 14, EGFR negative (non-basal-like). pN 1/1 (micrometastasis).

Our interpretation of the case, using the classification according to the site of tumour origin: Multifocal Grade 3 dedifferentiated DAB (3rd phase) combined with Grade 3 neoducts (2nd phase) over a region measuring 30x20 mm. The individual foci measured 12x10 mm, 11x10 mm and additional smaller satellites in the size range of 2 and 1 mm. IHC biomarkers: ER positive, PR negative, HER2 negative, Ki-67 24%, E-cadherin positive. CK 5/6, CK 14 and EGFR were all negative (non-basal-like). pN 1/1 (micrometastasis).

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Follow-up: Forty-five months after diagnosis and initiation of treatment, the patient was diagnosed with liver metastases. The combined evaluation of the histopathologic images and the IHC biomarkers confirmed that the metastases originated from the breast cancer (Figure 4).

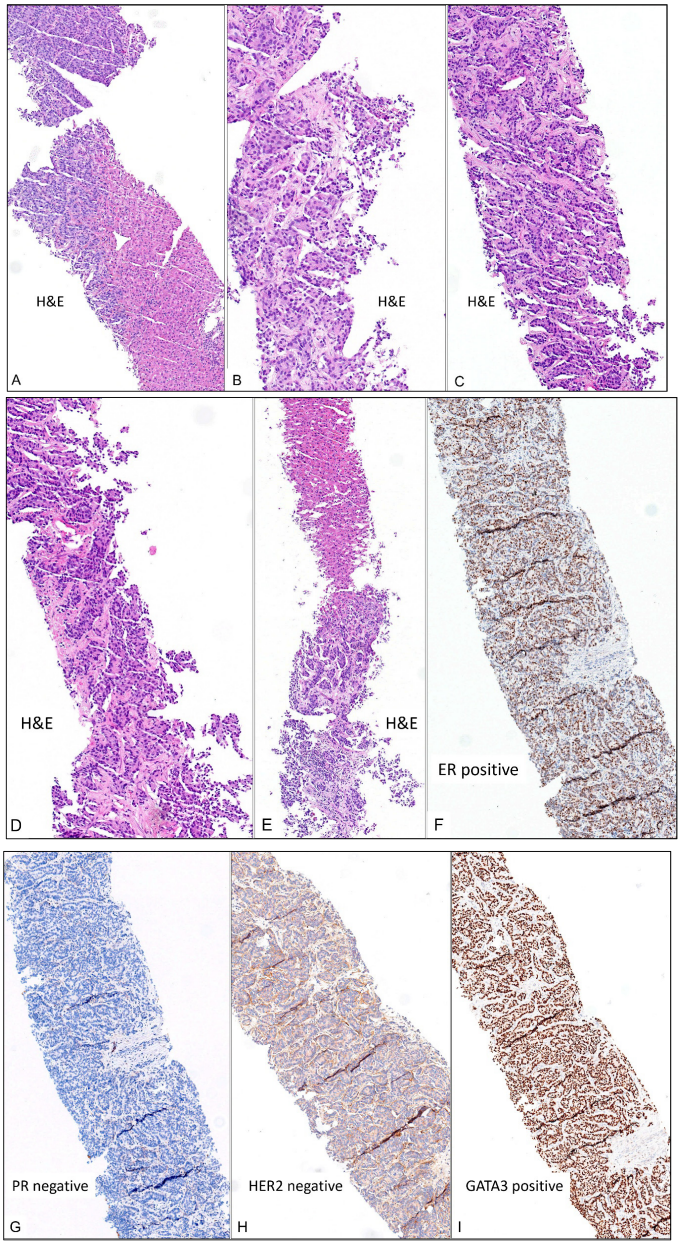


Figure 4 14G core biopsy samples of the liver metastases, H&E staining (A-E). The metastases were ER positive (F), PR and HER2 negative (G,H) and GATA3 positive (I), indicating the breast cancer origin of the metastases.

Outcome: Five years and five months after diagnosis and initiation of treatment the patient died from dedifferentiated DAB.

Second case

A 66-year-old woman detected a tumour in the upper-outer quadrant of her right breast. She had previously undergone bilateral breast reduction surgery, resulting in multiple regions of architectural distortion and numerous foci of posttraumatic type microcalcifications. These had remained largely unchanged during her previous three screening mammograms (Figure 5). Microfocus magnification images of the region corresponding to the palpable thickening and AI findings are shown in Figure 6. Following the imaging workup and ultrasound-guided 14G core biopsy (Figure 7) the patient underwent mastectomy (Figure 8) and was treated with chemotherapy and Herceptin, but not with radiotherapy or endocrine therapy.

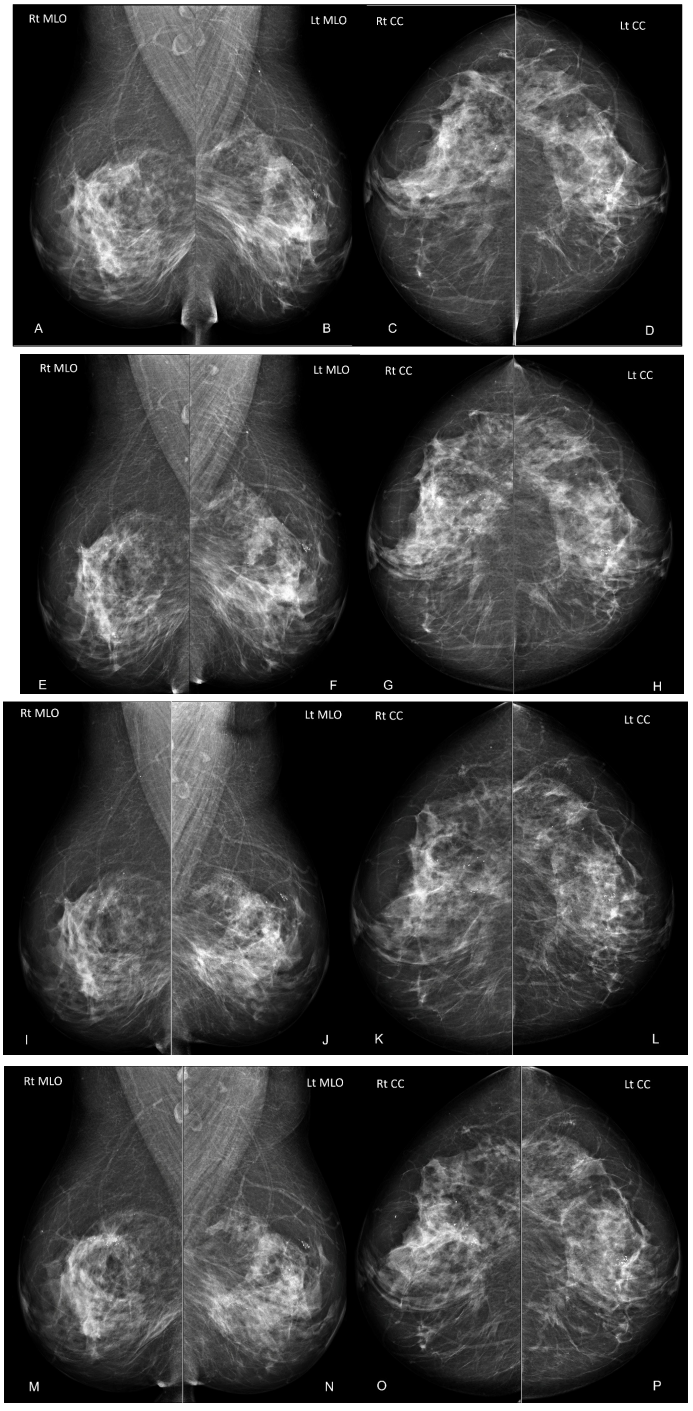
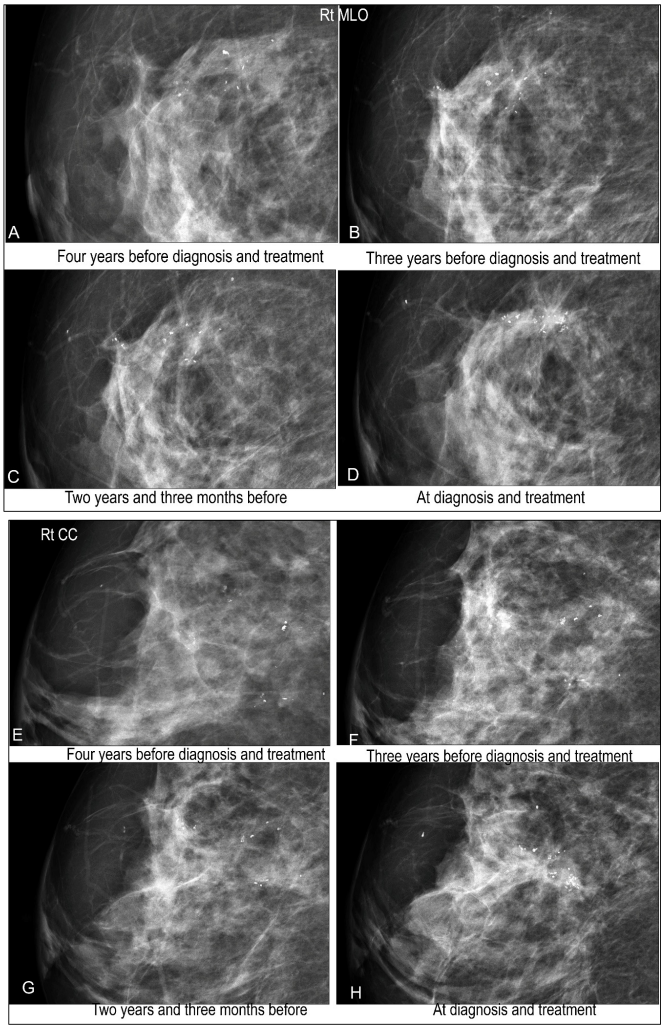


Figure 5 Four consecutive mammography examinations show the evolution of breast cancer. The time difference was eight months between the first (A-D) and second (E-H) screening examinations. The third examination (I-L) followed the second screening in 14 months. The time difference between the third and fourth examination (M-P) was 26 months, when the patient presented with a firm, palpable thickening in the upper-outer quadrant of her right breast. The excessive posttraumatic architectural distortion following breast reduction surgery is the most striking finding at each examination, both on the MLO and CC projections. An additional finding is clustered microcalcifications in both

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breasts. The radiologists considered them to be the consequence of bilateral breast reduction surgery, traumatic fat necrosis type calcifications.



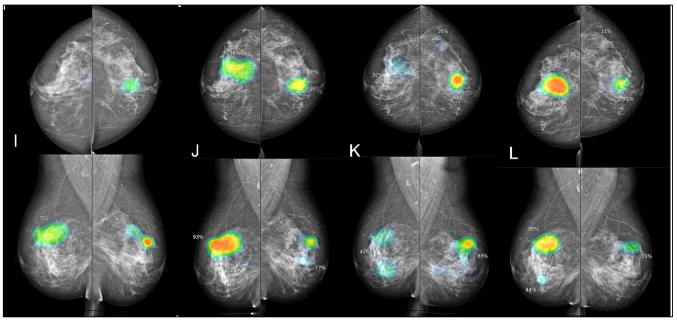
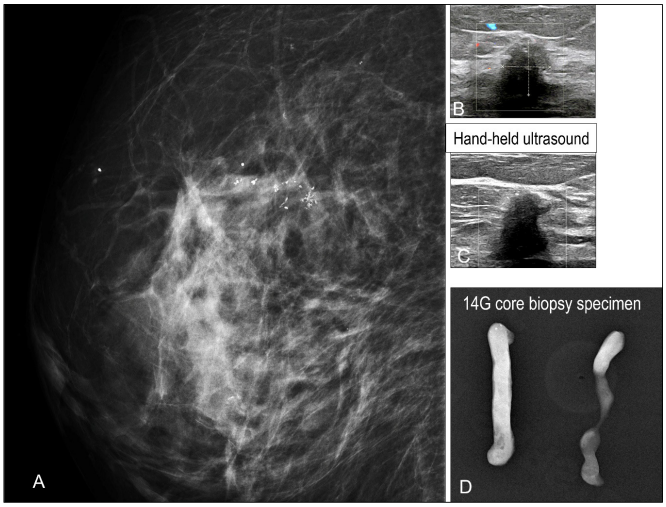


Figure 6 The microfocus magnification of the serial MLO projections (A-D) and the serial CC projections (E-H) show small variations in the appearance and distribution of the calcifications in the first three examinations. The appearance of malignant-type casting microcalcifications and an adjacent stellate tumour can be observed on the images of the fourth examination (D,H). The retrospective application of an artificial intelligence algorithm (I-L) marked all types of calcifications and scar tissue with varying degrees of reliability.



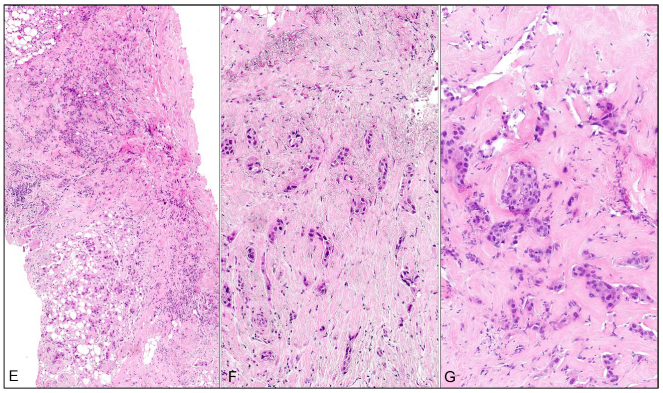
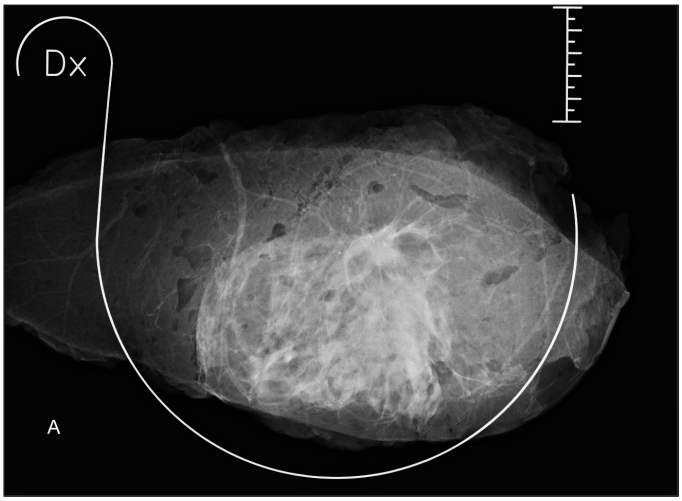
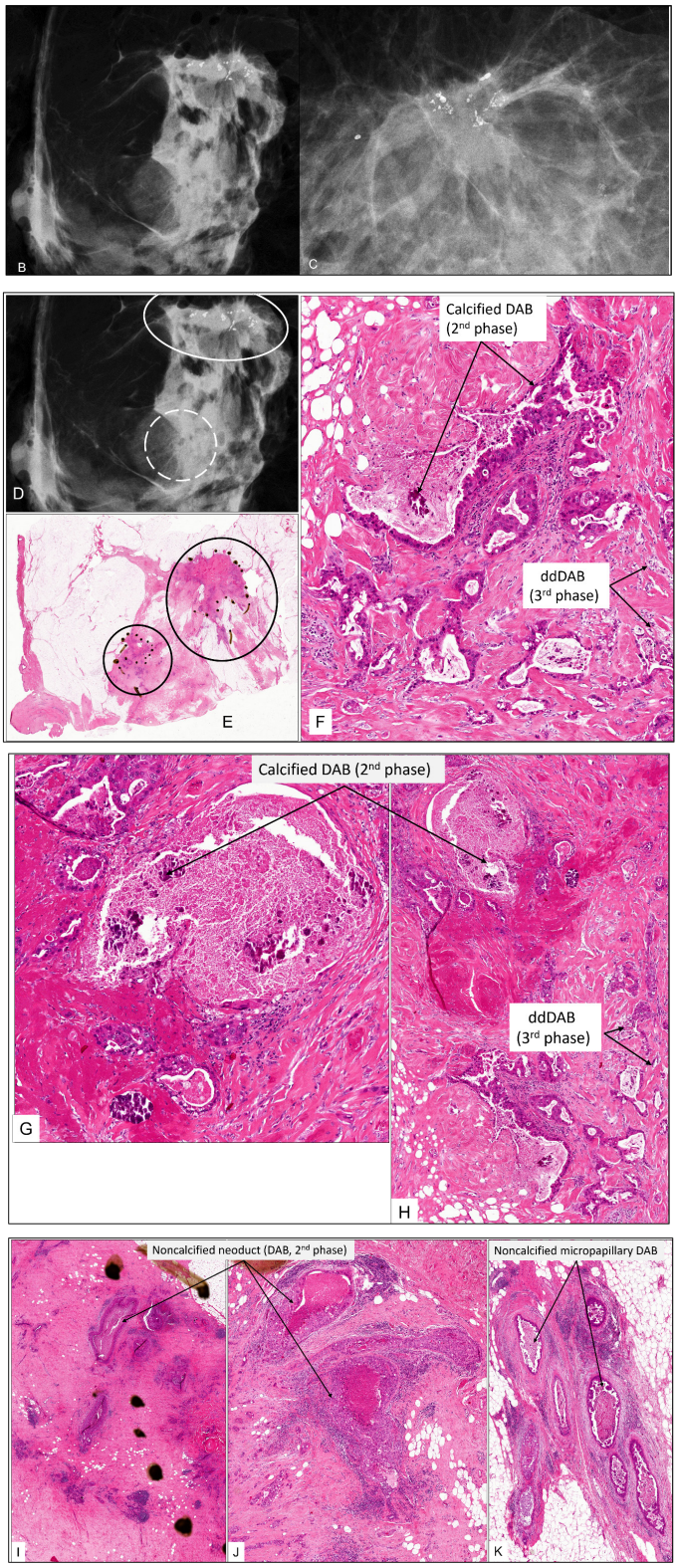


Figure 7 Right MLO microfocus magnification mammography (A) showing extensive architectural distortion associated with malignant type calcifications. Hand-held ultrasound (B) confirmed the presence of a malignant breast tumour. 14G core biopsy under ultrasound guidance (C) and core biopsy specimen radiograph (D). Histopathology specimens show both DAB and ddDAB in close proximity (E-G).



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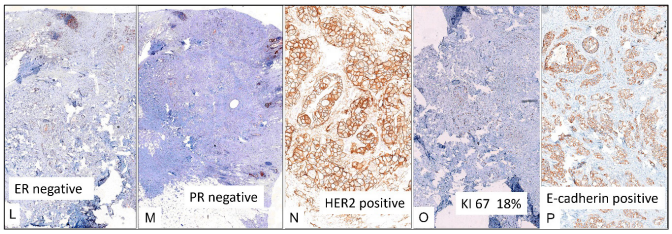
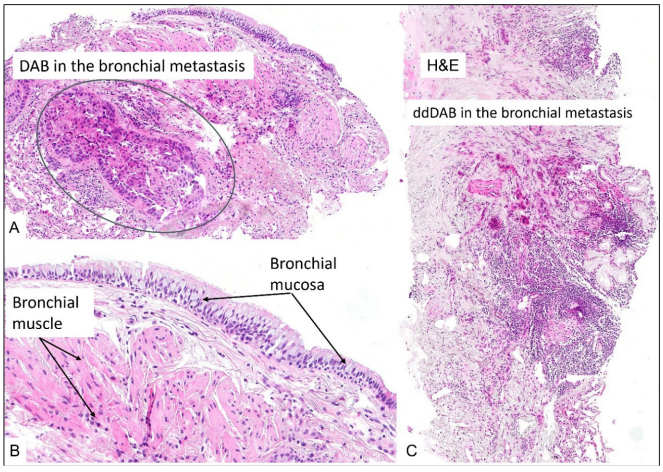


Figure 8 Mastectomy specimen radiograph (A). Specimen slice radiograph without microfocus magnification (B) and another with microfocus magnification (C) demonstrate the stellate tumour mass associated with malignant type calcifications. Mammographic (D) and large format thin section histopathologic (E) correlation reveals two invasive tumour foci (encircled in Figs. D and E). Intermediate-power histopathology images (F-K) richly illustrate the 2nd and 3rd phases of cancer originating within the major lactiferous ducts. Immunohistochemical biomarkers: ER (L) negative, PR (M) negative, HER2 (N) positive, Ki-67 (O) 18%, E-cadherin (P) positive.

The official histopathology diagnosis: Poorly differentiated bifocal invasive ductal carcinoma of apocrine type (20x17 mm and 10x7 mm) associated with 42x25 mm Grade 3 DCIS. Tumour-free margin: 10 mm. IHC biomarkers: ER/PR negative, HER2 positive, proliferation index (Ki-67) 18 %. Basal-like carcinoma (CK 5/6 positive, EGFR positive), E-cadherin positive. pN 0/1.

Our interpretation of the case, using the classification according to the site of tumour origin: Two foci of invasive dedifferentiated DAB (3rd phase) of apocrine type (20x17 mm and 10x7 mm) associated with 42x25 mm Grade 3 DAB (2nd phase). Tumour-free margin: 10 mm. IHC biomarkers: ER/PR negative, HER2 positive, proliferation index (Ki-67) 18 %. A tumour with basal marker expression (CK 5/6 positive, EGFR positive), E-cadherin positive. pN 0/1.

Follow-up: Twenty-eight months after diagnosis the patient was diagnosed with bronchial metastases (Figure 9).



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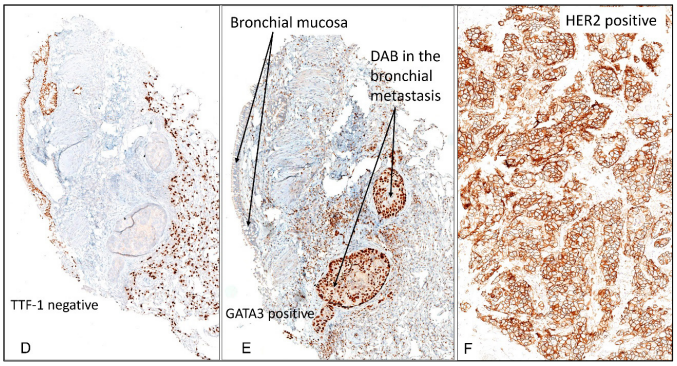


Figure 9 Histopathology of bronchial metastases of DAB and dedifferentiated DAB (A-C). IHC biomarkers: TTF-1 (lung and thyroid cancer marker) negative (D), GATA3 positive (E). DAB (2nd phase) in the bronchial metastasis is well illustrated with GATA3 staining (E). The strong TTF-1 staining (brown) shows the normal bronchial epithelium. This is the positive internal control and the tumour is TTF-1 negative. HER2 positive metastasis (F).

Outcome: Four years and four months after diagnosis and treatment, the patient died from metastasizing neoducts (DAB) and dedifferentiated DAB (ddDAB).

Discussion

In the process of searching for reasons why certain breast cancer subgroups are fatal despite our best efforts, our research group has correlated the multimodality imaging and large format thin and thick section histopathologic features with long-term follow-up data of 3,568 consecutive breast cancer cases.[1,7-10] Of these, 570 cases had their origin in the major lactiferous ducts. Our findings show that there is a considerable difference in the evolution and pathophysiology of breast cancers originating in the cell layer lining the major ducts, a complex three-phase process, compared with those cancers originating in the luminal cells of the acini of the terminal ductal-lobular unit, TDLU, a two-step process.[1] The earliest phase of both cancerous processes is *in situ*. When microcalcifications are present in cancers developing in the TDLUs, the imaging biomarkers are powdery, psammoma body-like or crushed stone-like, amorphous microcalcifications. Unfortunately, the current histopathologic terminology uses the term "DCIS" to describe this initial phase of cancer development within the TDLUs (lobules), which is a misleading misnomer. Instead, the initial phase of breast cancer arising within the TDLU is *in situ* acinar adenocarcinoma of the breast (*in situ* AAB) when taking the site of tumour origin into account.[4,8,9] The cancer progresses from this *in situ* phase to the 2nd, invasive phase. Histopathology terminology, as currently practiced, still uses the term "invasive ductal carcinoma" to describe this 2nd phase of evolution within the TDLU, while an anatomically more correct term would be invasive acinar adenocarcinoma of the breast (AAB), analogous to the term acinar adenocarcinoma of the prostate or parotid gland (AAP). The imaging biomarkers of this 2nd phase of AAB are stellate and/or ill-defined circular/oval-shaped tumour masses.[8,9]

The evolution of cancers originating from the epithelial cells lining the major lactiferous ducts is a more complex, three-phase process, as our investigations carried out over several decades have shown. The earliest phase of cancers of ductal origin can be considered a true "ductal carcinoma *in situ*" (DCIS), which is mammographically occult and very rarely detected at this early phase.[1,7] The next phase is a well-documented, remarkable duct-forming invasive process, the result of obvious structural dedifferentiation of the 1st phase. The newly formed, contorted, irregular duct-like structures (neoducts) invade the surrounding stroma, while the aggressive cancer cells remain confined within the neoduct borders, often surrounded by a thick periductal desmoplastic reaction (coated infiltration).[2-7] Neoducts can be differentiated from cancer-filled pre-existing ducts by their tortuous, contorted and tightly packed architecture, the absence of associated TDLUs, the periductal desmoplastic reaction with tumour-infiltrating lymphocytes, and periductal tenascin C overexpression.[1,4,5] The term *in situ* should be restricted to cancer confined to the patient's pre-existing ducts and lobules; by this definition newly formed duct-like structures (neoducts, 2nd phase) cannot be *in situ*, regardless of their boundaries. The current histopathology terminology uses the same "DCIS" term as in the 1st phase to describe this vastly different 2nd phase, which is best demonstrated on large format thin and thick section histopathology images.[1]

Further progression of the structural dedifferentiation results in the escape of the cancer cells through defects in the integrity of the boundaries of the neoducts.[1] This was described as early as 1953 by Willis.[10] The aggressive cancer cells proliferate within the interstitial space of the periductal stroma and can disseminate to distant organs.[1,11] It is important to note that the dynamic change affects the structure but not to the epithelial cells. The poor outcome of these patients cannot be attributed to "microinvasion" because the 3rd phase is a structurally dedifferentiated tumour cell population that has lost the ability to form duct-like structures. Our proposed three-phase evolution accounts for the close association and coexistence of the 2nd and 3rd phases. The importance of diagnosing both in the same specimen was also known by Willis in 1953 when writing "…the pathologist must examine many parts of the diseased breast in search for invasive growth, for it is the presence or absence of this which will determine the prognosis".[10]

The current terminology calling the 2nd phase "DCIS" constrains research to evaluate the use of Herceptin treatment at an earlier phase of this aggressive disease subgroup, when it could prevent or delay progression through structural dedifferentiation to the 3rd phase. The CAP Consensus Statement maintains that "Only the invasive component of a tumour (not *in situ* disease) should be scored.".[12-15] Although the NSABP B-43 trial tested HER2 in "DCIS" and randomized patients to Herceptin, there is a considerable difference between the 2nd phase of our three-phase theory and the "DCIS" cases in the NSABP B-43 trial, where the CIS AAB cases were included as DCIS.[16] Neither of the present cases can answer this question: Case 1 was HER2 negative, and Case 2 received Herceptin only after the 3rd phase had developed. Whether HER2-targeted treatment of the 2nd phase can prevent or delay progression can only be answered by a prospective study.

Conclusion

The site of origin of breast cancer is of primary importance to each patient's prognosis. Possession of a more realistic prognostic evaluation can enhance our ability to provide a more favourable outcome. The use of imaging biomarkers helps us distinguish the sites of tumour origin and can also help predict the efficacy of the current therapeutic tools; they are considerably more successful in those tumours that have their origin in the TDLUs than in cancers originating in the major lactiferous ducts. Our proposed theory of three phases of evolution considers the 2nd phase to be an invasive carcinoma of stem cell origin,[17] suggesting the need for appropriate treatment for this diffusely invasive disease, including the need for extensive surgical intervention and research regarding the benefit of using Herceptin in this earlier phase.

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Acknowledgements

The authors are greatly indebted to Professor F. Lee Tucker for his contribution to the conception of the process of structural dedifferentiation. Special thanks to Ms. Helena Hermelin, the leader of the Research Laboratory at Falun Central Hospital, for high quality scanning the histopathology glass slides and organizing the research archive. This work has been supported by funding from the American Cancer Society through a gift from the Longaberger Company's Horizon of Hope® campaign (Project NHPDCSGBR-GBRLONG).

Ethics Committee permission 2022-10-18: Dnr 2022-05366-02

Conflicts of interest

The authors declare that they have no competing interests.

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