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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 II

Abstract

Background: Breast cancers originating in the epithelia of the major ducts develop through three steps of progression, the 1st being a mammographically occult *in situ* phase. In the 2nd phase neoducts branch out from the original duct(s) forming an invasive network through the process of neoductgenesis. Neoducts are frequently surrounded by a desmoplastic reaction and lymphocytic infiltration, producing a duct forming invasive carcinoma, termed ductal adenocarcinoma of the breast (DAB). In the 3rd phase defects in the neoduct boundary develop through structural dedifferentiation, allowing the malignant cells to disseminate freely beyond these restrictive neoducts. This 3rd step is associated with a vastly increased fatality and is termed dedifferentiated ductal adenocarcinoma of the breast (ddDAB).

Precis: These case descriptions provide additional, richly illustrated examples of neoductgenesis and structural dedifferentiation of cancers originating within the major lactiferous ducts. The lack of significant progress in reducing the fatality of this group of breast cancers can be attributed to a poor understanding of their complex nature.

Case description: Case 1 describes a 48-year-old woman who detected a firm thickening in the upper portion of her left breast five years after her most recent, negative mammograms. Multimodal imaging shows a five cm region having fragmented casting type calcifications associated with a non-specific asymmetric density. Correlation of imaging and large format thin section histopathology demonstrated an extensive malignancy of ductal origin (DAB) associated with multifocal invasive dedifferentiated DAB. Four years after diagnosis and initiation of treatment the patient was diagnosed with distant metastases in the sternum and in the lungs. The distant metastases had histopathologic and immunohistochemical features similar to those of the malignant neoducts (DAB) and dedifferentiated DAB in the breast. Four years and six months after diagnosis and treatment the patient died from metastasizing DAB and ddDAB.

Case 2 describes a 52-year-old woman who suffered an unfortunate scenario which is often typical for this breast cancer subtype represented by numerous fragmented type calcifications on the mammogram and by the rapid development of an extensive breast malignancy. The first symptom of this patient was shortness of breath caused by pleural metastases. A search for the primary tumour detected a firm, palpable, diffuse thickening in the upper-outer portion of the right breast. Mammograms showed many fragmented casting type calcifications between the nipple and the chest wall, a characteristic imaging biomarker of breast cancer originating within the major lactiferous ducts. The patient was inoperable, but the microscopic diagnosis was confirmed by 14-G biopsy, which also provided a sufficient amount of tissue to evaluate the IHC biomarkers on both the core biopsy specimen as well as the tissue samples of the pleural metastasis. The patient died from metastasizing dedifferentiated DAB eleven months after diagnosis and initiation of palliative treatment.

Conclusion: Current histopathologic terminology and practice considers that the 1st and 2nd phases in the evolution of breast cancers originating within the major lactiferous ducts are identical by using the same term to describe these two morphologically distinct entities as "DCIS". This interpretation fails to account for the presence of DCIS-like structures within axillary lymph nodes and in vital organs, or why "microinvasion" is associated with extensive distant metastases and accounts for breast cancer death. The three phases of development proposed by our research group form a fundamental concept supporting efforts to control this aggressive breast malignancy subgroup characterized by fragmented casting type calcifications associated with structural dedifferentiation of neoducts.

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

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László Tabár,[1] Peter B Dean,[2] Miklós Tarján[3]
[1]Department of Mammography, Falun Central Hospital, Sweden
[2]Department of Diagnostic Radiology, Faculty of Medicine, University of Turku, Finland
[3]Department of Clinical Pathology and Cytology, Falun Central Hospital, Sweden

Correspondence: László Tabár, Department of Mammography, Falun Central Hospital, Sweden

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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 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, glutamyl aminotransferase subunit A transcription factor 3; CK 7, cytokeratin 7 staining; TTF-1, thyroid transcription factor 1

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Introduction

The anatomic site of origin of a malignancy has a profound influence upon its development and outcome. Breast cancers arising within the major lactiferous ducts have a characteristic course of evolution documented by correlating their imaging biomarkers (mammographic appearance) with large format histopathology and long-term clinical outcome.[1–6] Image assisted large section histopathology of excised breast tissue enables comprehensive examination of an approximately tenfold larger contiguous tissue area than is provided by conventional small section technology. A considerably improved documentation of breast cancer and a more reliable assessment of tissue margins is provided by using larger sized histopathology samples to correlate with breast imaging findings. These in turn can enable more appropriate treatment planning, improved surgical performance, fewer recurrences, and better patient outcome. Uncertainty of surgical margin evaluation inherent to the standard small section technique can lead to inappropriate decisions in surgical management and adjunctive therapy.[7]

Breast malignancies arising within the major lactiferous ducts develop initially as mammographically occult intraductal (*in situ*) tumours, which progress to a 2nd phase through the process of neoductgenesis, in which newly formed duct-like structures (neoducts) continuously branch out and expand (Figure 1A). The most characteristic subgroup has a solid tumour growth pattern associated with central necrosis, within which the pathognomonic casting-type calcifications (BI-RADS fine linear branching calcifications) develop, making the malignancy detectable on the mammogram (Figure 1B).[8,9] The invasive nature of these neoducts, documented by their presence in breast cancer metastases as early as 1967 and subsequently reported by several research groups,[5,10–16] has yet to be officially endorsed by the pathology community.[17] The current histopathologic term DCIS is misleading, since neoducts can spread to axillary lymph nodes and to distant organs.[3–6] (Figure 2)

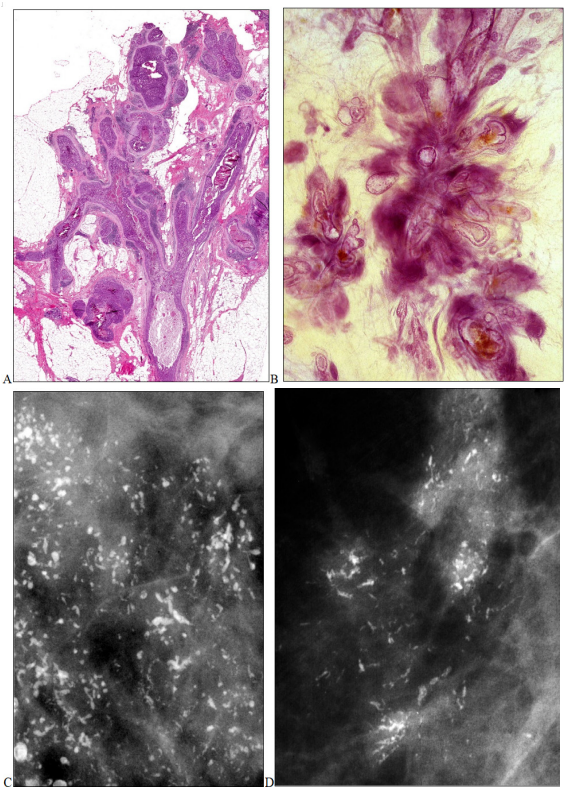


Figure 1 Large format thin (A) and subgross, thick (B) section histopathologic images of neoducts (DAB), the 2nd phase in the development of cancer originating within the major lactiferous ducts. Although the current histopathology uses the term DCIS, these tightly packed, contorted, newly formed duct-like structures lack associated TDLUS and do not resemble of the patient's pre-existing ducts, making the term ductal carcinoma *in situ* (DCIS) a misleading misnomer. The mammograms (C,D) reflect the underlying histopathology image showing numerous fragmented casting-type calcifications.

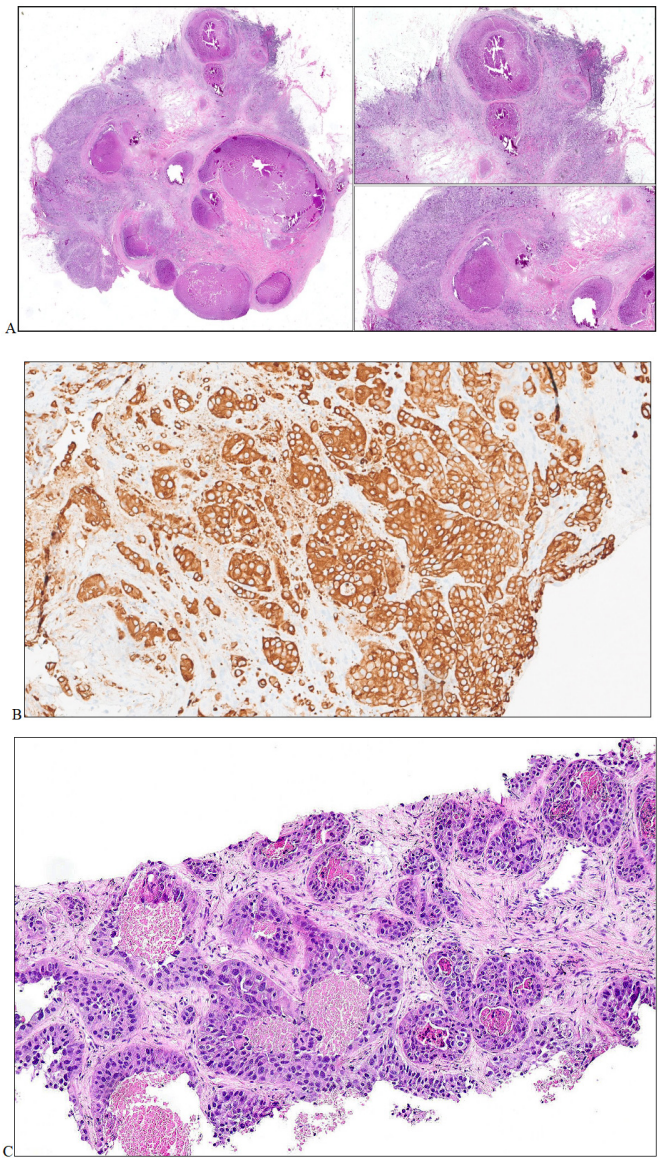


Figure 2 Neoducts (DAB) are demonstrated in multiple axillary lymph nodes (A) and in bone (B) (cribriform DAB metastasis in the sternum, Cytokeratin AE1-AE3 immunohistochemical staining, see Case 1, Fig. 7C), as well as in lung metastases (C).

In the 3rd phase of progression, the neoduct boundary undergoes structural dedifferentiation when breaches develop in the boundary of the neoduct through which the tumour cells are able to escape to the periductal tissue and to the interstitial spaces, where they propagate, forming unifocal or multifocal tumours that can metastasize (Figure 3).[5,6]

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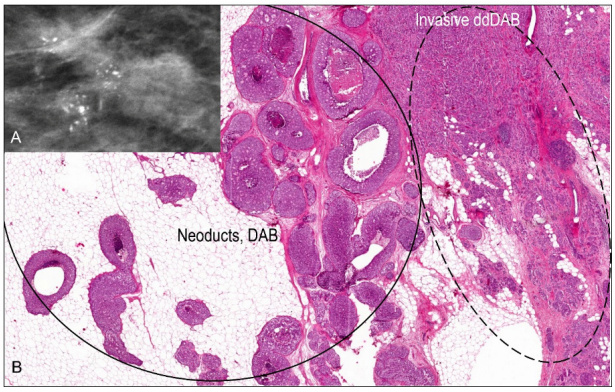
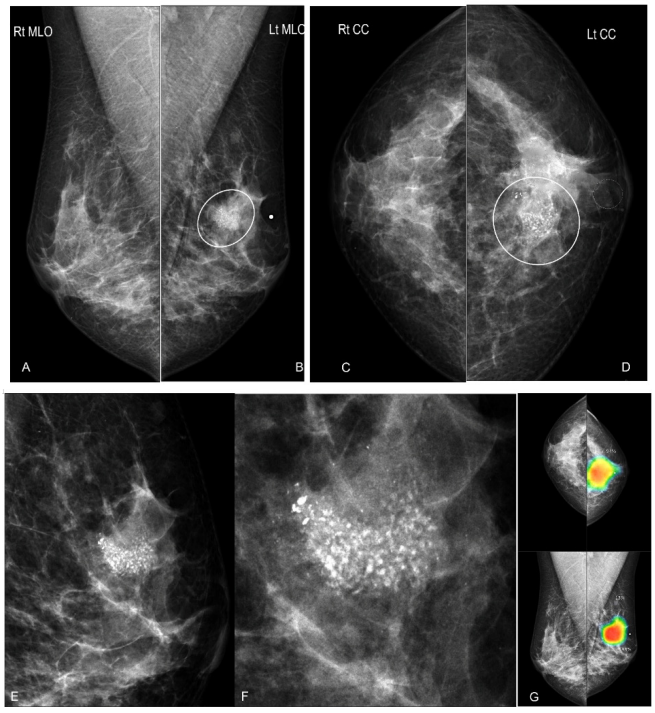


Figure 3 Mammographic (A) and large format histopathologic (B) correlation of DAB (encircled, solid line) and dedifferentiated DAB (encircled, dashed oval).

This report is based upon a comprehensive series of breast cancers described in three previous publications.[4–6] Both cases have typical casting-type calcifications on the mammograms, the imaging biomarker of neoductgenesis.[9]

First case

A 48-year-old woman with a self-detected firm thickening in the upper portion of her left breast five years after her previous, negative mammogram. Multimodality imaging workup revealed numerous fragmented casting type calcifications over a region measuring five centimetres, corresponding to the palpatory finding in the left breast (Figure 4). Low and higher power, large format histopathology images correlated with imaging details (Figure 5).



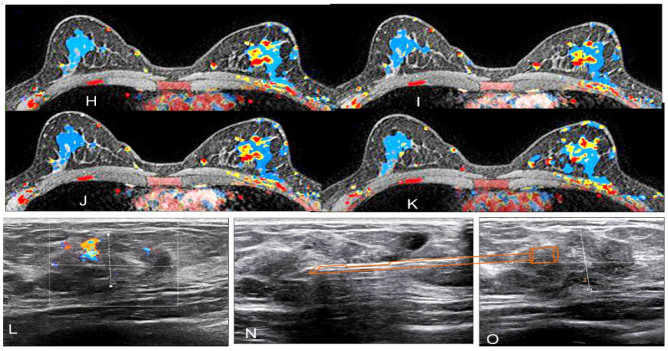
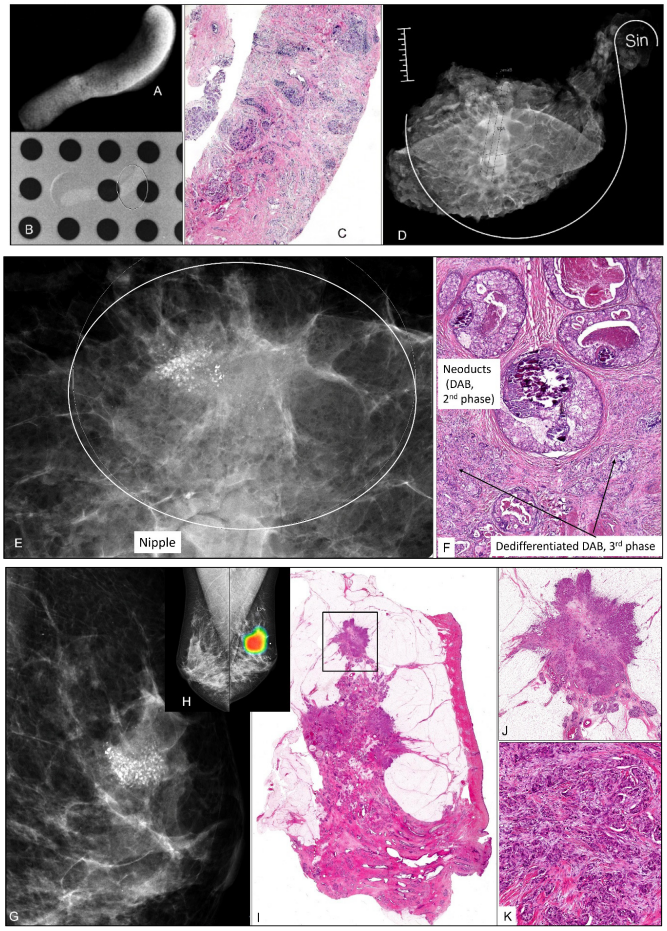


Figure 4 MLO (A,B) and CC (C,D) projections. Normal right mammograms. The asymmetric density in the upper-mid portion of the left breast (encircled) contains microcalcifications. Latero-medial horizontal projection (E) and microfocus magnification (F) show a large group of malignant type, fragmented casting type calcifications. Artificial intelligence algorithm (G) marked the abnormal region. A series of MRI images (H-K) demonstrate the extent of the disease and several foci having increased enhancement. Hand-held ultrasound with Doppler and US-guided 14G core biopsy (L-O).



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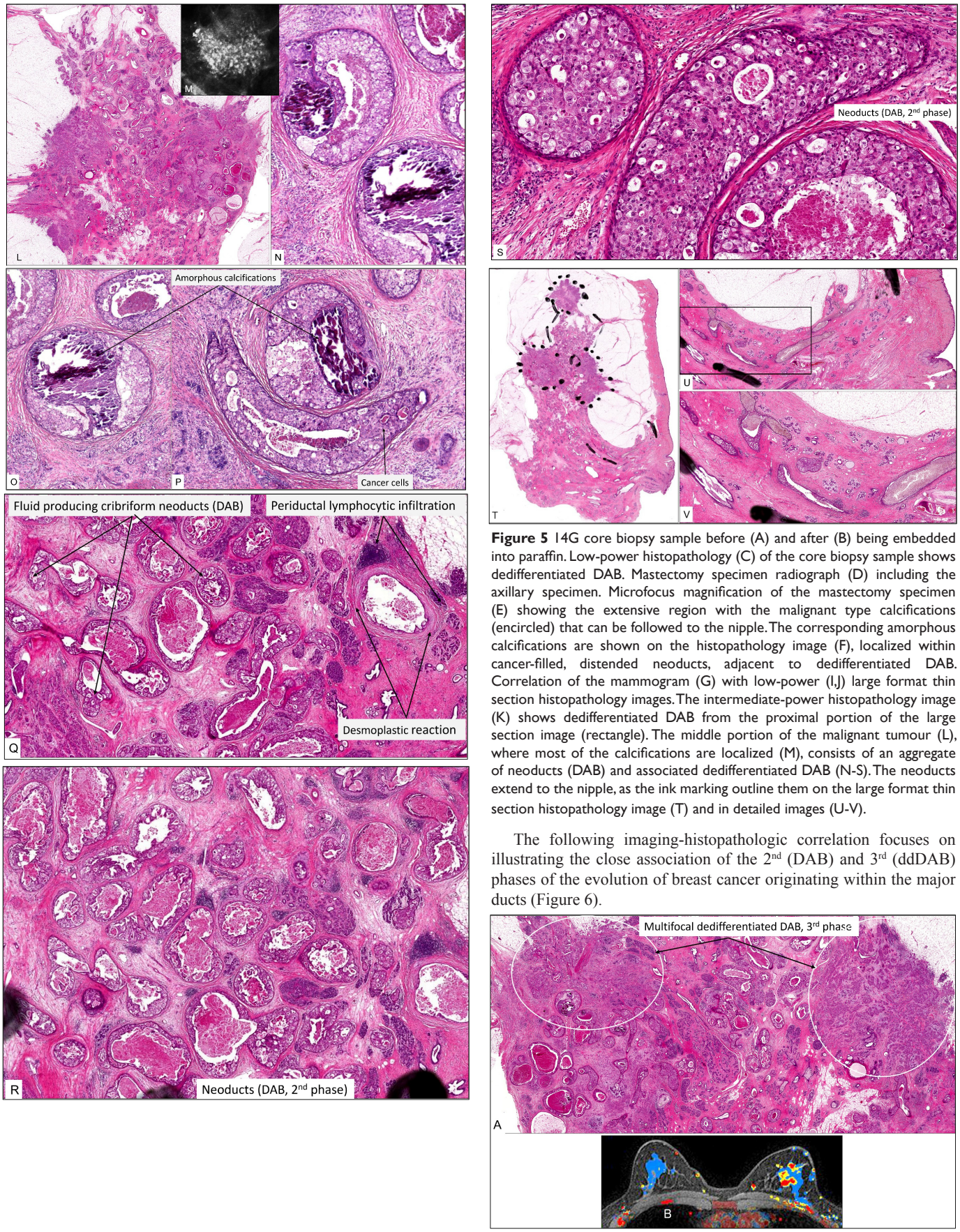


Figure 5 14G core biopsy sample before (A) and after (B) being embedded into paraffin. Low-power histopathology (C) of the core biopsy sample shows dedifferentiated DAB. Mastectomy specimen radiograph (D) including the axillary specimen. Microfocus magnification of the mastectomy specimen (E) showing the extensive region with the malignant type calcifications (encircled) that can be followed to the nipple. The corresponding amorphous calcifications are shown on the histopathology image (F), localized within cancer-filled, distended neoducts, adjacent to dedifferentiated DAB. Correlation of the mammogram (G) with low-power (I,J) large format thin section histopathology images. The intermediate-power histopathology image (K) shows dedifferentiated DAB from the proximal portion of the large section image (rectangle). The middle portion of the malignant tumour (L), where most of the calcifications are localized (M), consists of an aggregate of neoducts (DAB) and associated dedifferentiated DAB (N-S). The neoducts extend to the nipple, as the ink marking outline them on the large format thin section histopathology image (T) and in detailed images (U-V).

The following imaging-histopathologic correlation focuses on illustrating the close association of the 2nd (DAB) and 3rd (ddDAB) phases of the evolution of breast cancer originating within the major ducts (Figure 6).

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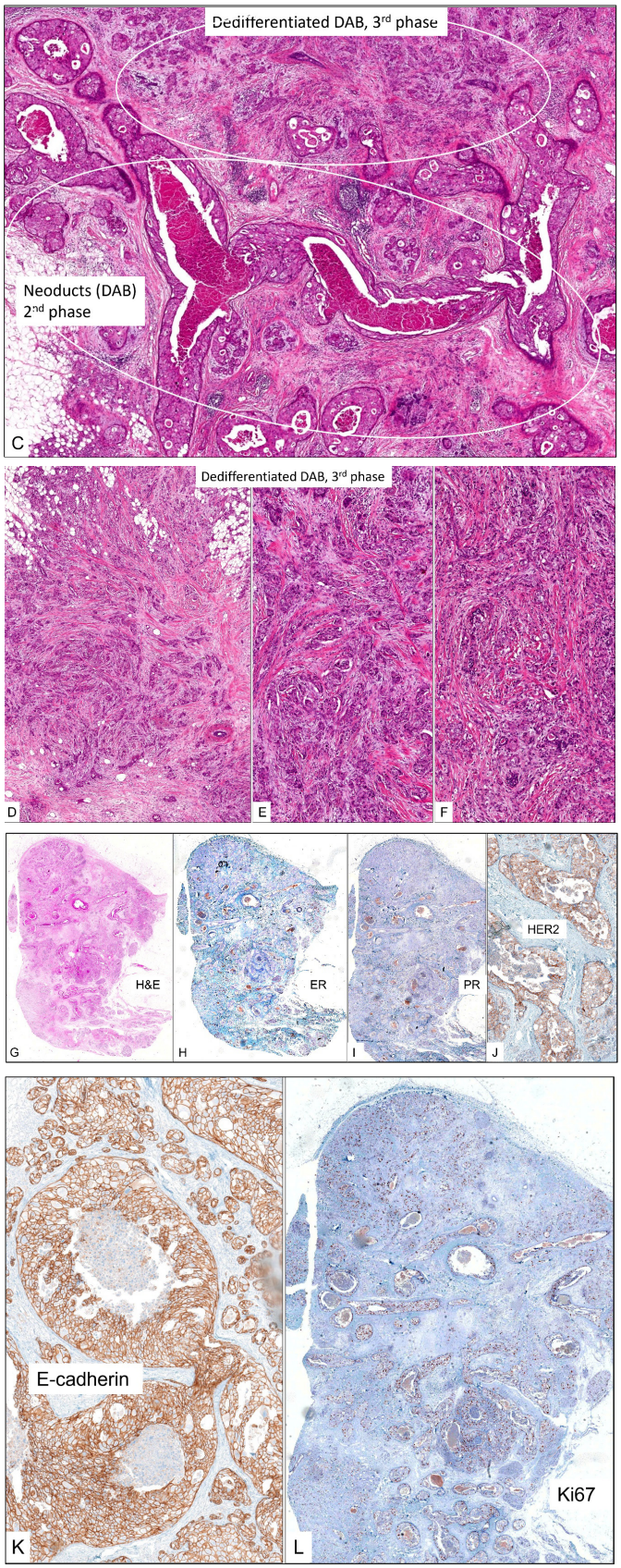
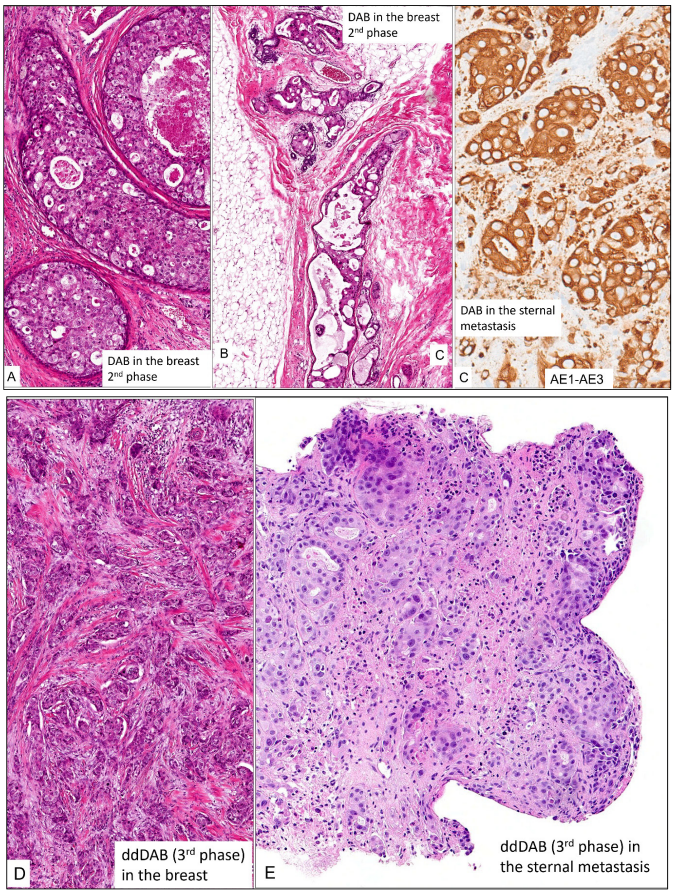


Figure 6 Breast MRI (B) demonstrates the extent of the disease. The enhancement corresponds to the multiple dedifferentiated DAB foci. The large format thin section histopathology images (A,C) illustrate the coexistence of extensive neoducts (DAB) and multifocal dedifferentiated DAB foci. Higher power histopathology images of the dedifferentiated DAB are seen on separate images (D-F). H&E staining (G). Immunohistochemical biomarkers, ER/PR/HER2 negative (H,I,J) indicate a triple negative tumour. E-cadherin positive (K), Ki67 (L) (6%).

The official histopathology diagnosis: Multifocal, moderately differentiated invasive apocrine breast cancer. The individual foci measured 15x8 mm, 13x10 mm, 10x10 mm, associated with 50x30 mm Gr 3 apocrine DCIS. Minimal (1 mm) tumour-free margin toward the chest wall. pN 0/13. IHC biomarkers: triple negative tumour, ER/PR/HER2 negative, E-cadherin positive, Ki 67 shows mitotic activity in 6% of the tumour cells. EGFR positive.

Our interpretation of the case: Multifocal invasive dedifferentiated ductal adenocarcinoma of the breast (ddDAB) associated with 50x30 mm Gr 3 neoducts (DAB). The individual foci measured 15x8 mm, 13x10 mm, 10x10 mm. Minimal (1 mm) tumour-free margin toward the chest wall. pN 0/13. IHC biomarkers: triple negative tumour, ER/PR/HER2 negative, E-cadherin positive, Ki67 shows mitotic activity in 6% of the tumour cells. EGFR positive.

Follow-up: Four years after diagnosis and initiation of treatment the patient was diagnosed with metastases to the sternum (Figure 7).



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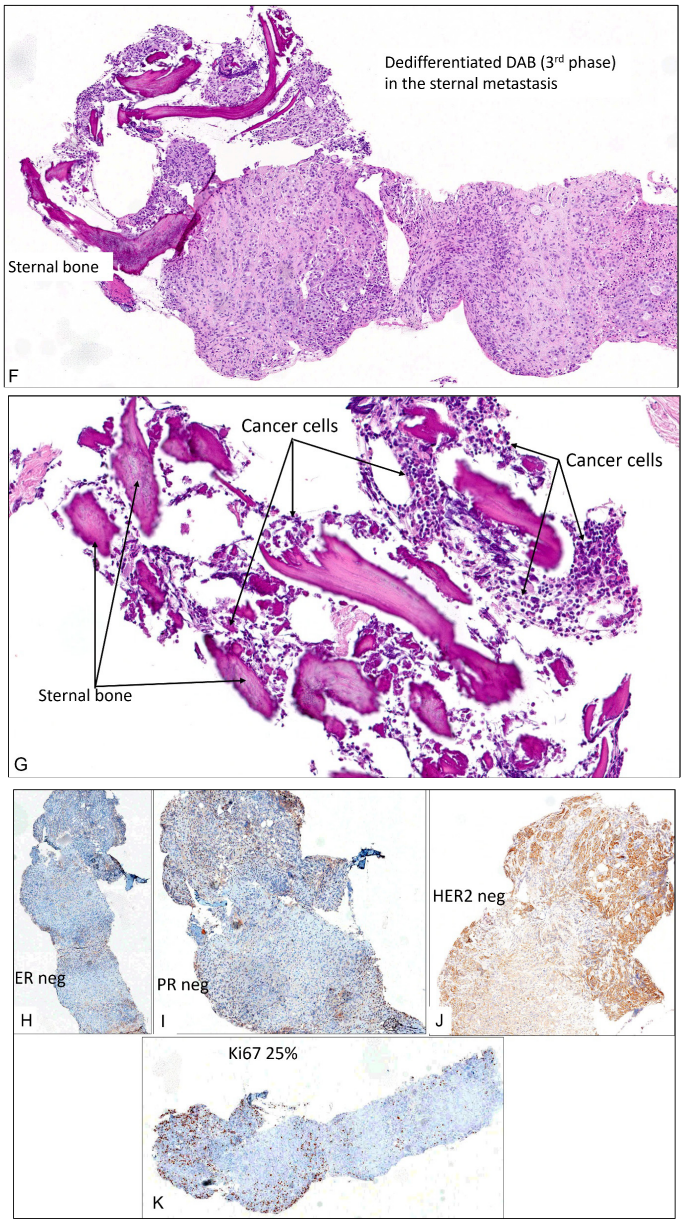


Figure 7 Demonstration of the histopathologic image of cribriform DAB (2nd phase) in the primary breast tumour (A,B) and a similar sternal metastasis (C). Dedifferentiated DAB in the primary breast cancer (D) is compared with the similar dedifferentiated DAB in the sternal metastasis (E). Bone fragments from the sternal biopsy are mixed with loose cancer cells (F,G). The immunohistochemical biomarkers of the sternal metastases (H-K) show a pattern similar to the IHC biomarkers of the primary tumour (triple negative cancer). Both histopathology and the IHC biomarkers confirm breast cancer metastasis to the sternum.

Further follow-up: Four years after diagnosis and initiation of treatment the patient was also diagnosed with bronchial metastases, diagnosed in the same week as the sternal metastases (Figure 8).

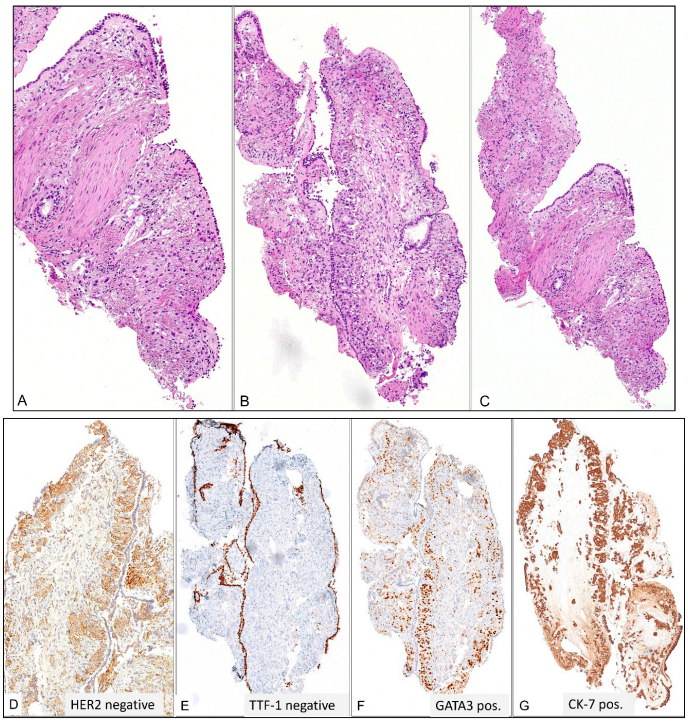


Figure 8 Invasive carcinoma in the subepithelial stroma was covered by normal bronchial respiratory epithelia cells (A-C). The metastases were TTF-1 (marker for lung and thyroid gland tumours) negative, GATA3 (breast cancer marker) positive, HER2 negative and CK-7 positive. Histopathology and the IHC biomarkers confirmed breast cancer metastasis to the bronchi.

Outcome: Four years and six months after diagnosis and initiation of treatment the patient died from metastasizing neoducts (DAB) and dedifferentiated DAB.

Second case

A 52-year-old woman was referred for mammography examination following the microscopic examination of a pleural effusion, suggesting that the primary tumour was breast cancer. She had attended mammography screening examinations regularly, most recently 23 months earlier. This case demonstrates the sudden emergence of this extensive, diffuse metastatic breast malignancy following a normal mammogram (Figure 9). The primary symptom was shortness of breath caused by distant metastases. The imaging-histopathologic correlation revealed both the 2nd (DAB) and 3rd (ddDAB) phase of the evolution of breast cancer originating within the major lactiferous ducts. The main mammographic sign was fragmented casting type calcifications, the imaging biomarker characteristic for this breast malignancy subgroup (Figure 10). Multimodality imaging workup revealed the very extensive nature of the disease originating within the major lactiferous ducts (Figure 10). The results of the microscopic examination of the 14G biopsy specimen and the immunohistochemical biomarkers are shown on Figure 11. The patient was diagnosed with pleural metastases immediately prior to her breast cancer diagnosis (Figure 12).

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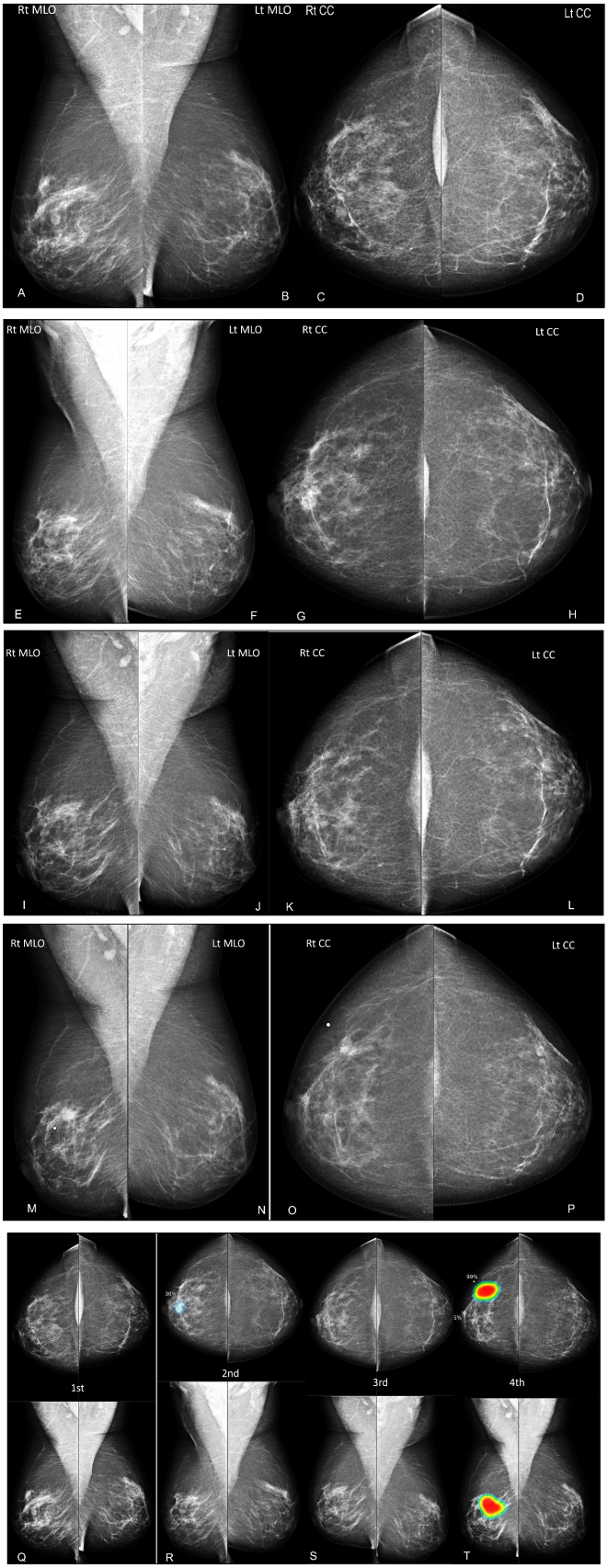
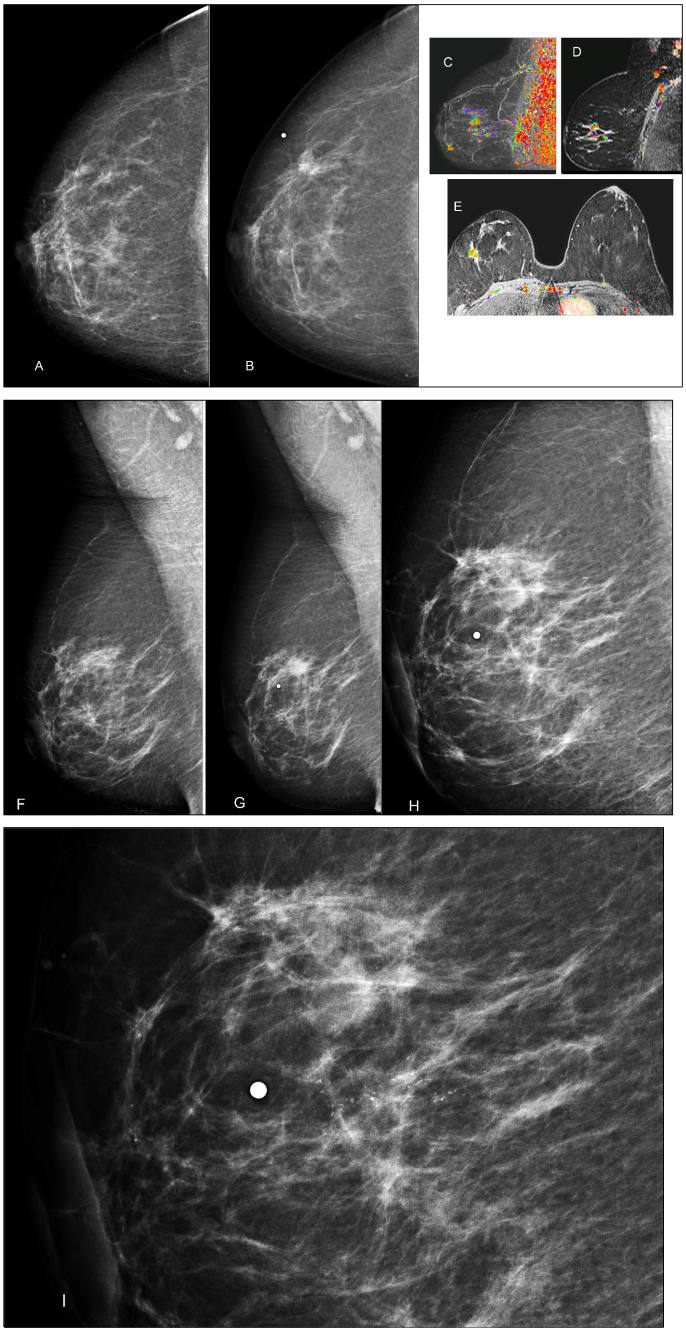


Figure 9 Four consecutive mammography screening examinations, MLO (A,B,E,F,I,J,M,N) and CC projections (C,D,G,H,K,L,O,P). The time difference was 14 months between the earliest examination shown here (A-D) and the second examination (E-H); the time-interval between the second and third examination (I-L) was 20 months and 23 months between the third and fourth examination (M-P). The patient presented with a palpable thickening in the upper-lateral portion of the right breast (M-P) at the fourth examination. Clinical breast examination confirmed the presence of diffuse, firm thickening in the upper-lateral portion of the right breast and one palpable pathologic lymph node in the right axilla. Artificial intelligence (Q-T) algorithm showed no definite malignancy earlier than at the fourth examination, when the lesion was already palpable.



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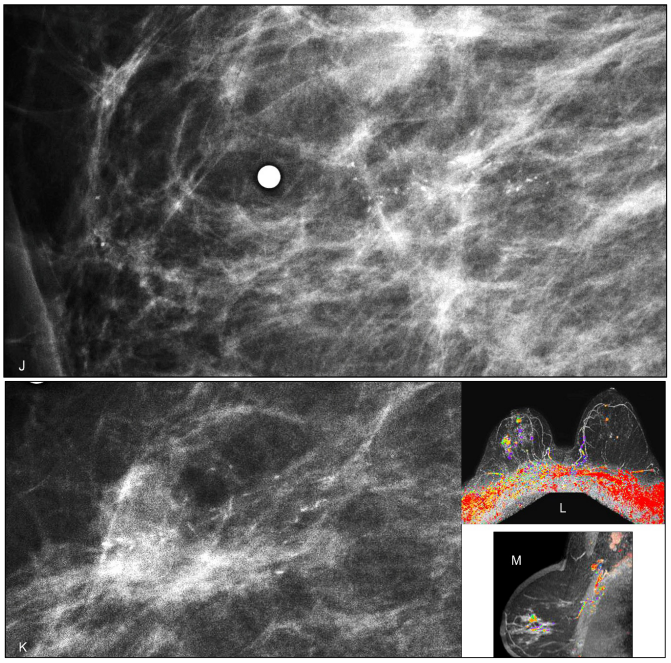


Figure 10 Comparison of the CC (A) and MLO (F) mammograms of the right breast from the third (previous) examination with the CC (B) and MLO (G) diagnostic mammograms at the time of her symptoms. Breast MRI (C-E) and the right MLO microfocus image (H) show diffuse disease occupying an entire lobe. Detailed microfocus magnification images (I-K) revealed many fragmented casting type calcifications between the nipple and the chest wall. Breast MRI images (L,M) confirm the diffuse distribution of the malignant disease.

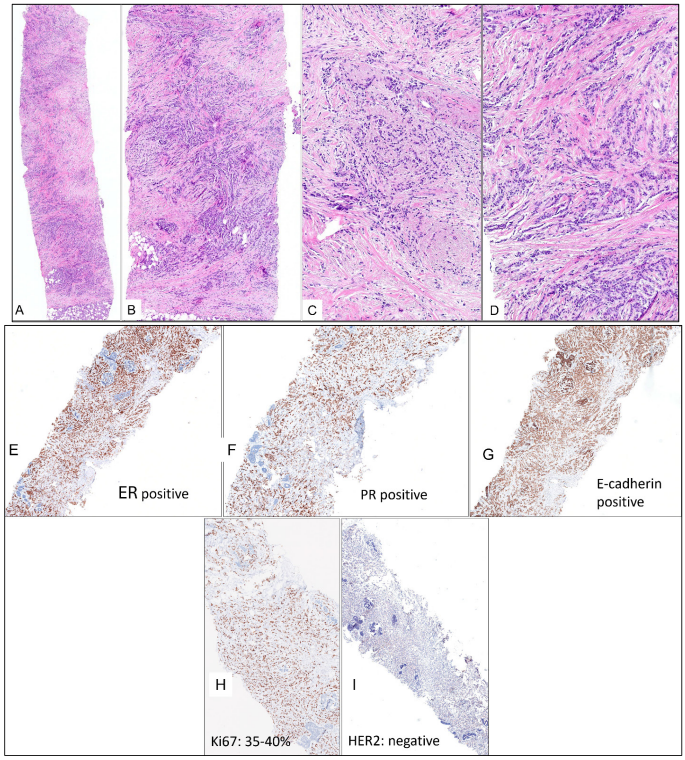


Figure 11 Low and higher power histopathology images of the 14G core specimen (A-D) show dedifferentiated DAB (3rd phase in the evolution of the cancer developing within the major lactiferous ducts). The tumour was ER/PR/E-cadherin positive and HER2 negative. The proliferation index (Ki67) was high (35-40%).

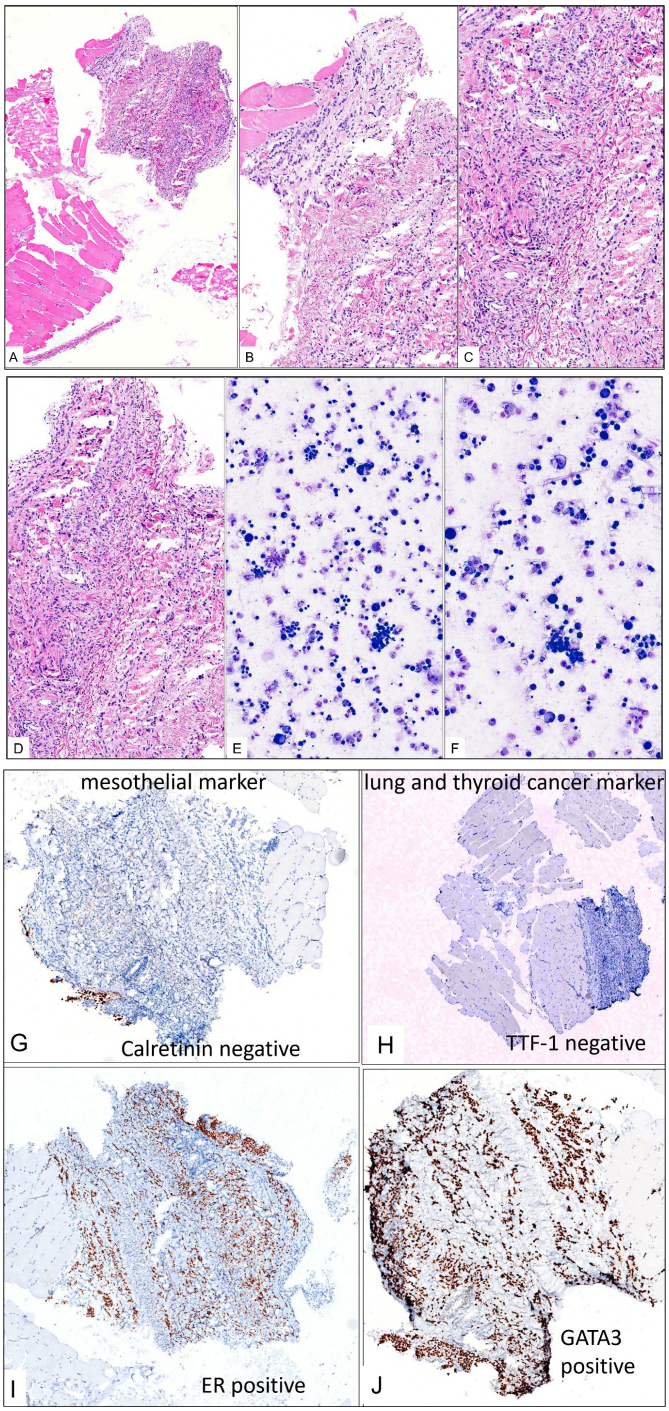


Figure 12 H&E staining of the pleural biopsy tissue samples (A-D) shows cancer cells comparable with the dedifferentiated DAB cancer cells in the primary breast cancer. Malignant cells are seen in the pleural effusion (E,F). The immunohistochemical biomarkers fully support the microscopic examination results: Calretinin (mesothelial marker) (G) negative, TTF-1 (lung and thyroid cancer marker) (H) negative, ER positive (I) and GATA3 (breast cancer marker) positive (J).

Outcome: The patient was considered to be inoperable. Eleven months after diagnosis and initiation of treatment the patient died from metastasizing dedifferentiated DAB (3rd phase of a cancer originating within the major lactiferous ducts).

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Discussion

Early detection of breast cancer through regular attendance in mammography screening and the use of modern therapeutic regimens have resulted in a significantly decreased breast cancer death up to 50%.[18–22] Approximately 20% of the remaining fatal cases result from the diffuse breast malignancies originating within the major lactiferous ducts.[23] While early detection combined with the use of modern therapy provided significant improvement in long-term survival over two successive epochs of breast cancers originating in the terminal ductal lobular units, no such improvement was observed in cancers originating within the major lactiferous ducts.[5]

Thorough imaging-large format histopathologic correlation of 317 consecutive breast cancers with fragmented casting type calcifications on the mammograms led us to realize that this breast malignancy evolves through three phases of development.[5] This progression does not involve cellular changes, since the malignancy grade is initially high in this cancer of stem cell origin; rather, it involves structural dedifferentiation during the progression of the disease, which can be objectively determined. Our large format thin and thick section histology glasses, measuring up to 10x8 cm of contiguous breast tissue, provide an objective answer to this question.

1) The normal, pre-existing major lactiferous duct measures 100 µm (0.1 mm). The gradual accumulation of cancer cells in the 1st phase of evolution makes the duct distend up to 20-30 times its normal diameter, while the architecture of the pre-existing, normal anatomic structure is still retained.

2) The 2nd phase is characterized by new duct formation, which is visually characterized by the increased number of haphazard, tortuous, duct-like structures, each of which is distended by cancer cells, central necrosis and occasional amorphous calcifications, all forming a convoluted mass-like structure that can occasionally be palpable. There is an expression for this in histopathology terminology: ”tumour forming DCIS.” The considerable deformation of the normal anatomic structure in the second phase is objectively measurable by a calliper for up to several centimetres.

3) In the third phase, when the extraductal cancer cells eventually form a stellate or circular tumour mass, the accumulation of cancer cells initially measures only a few millimetres, at which point pathologists describe it as ”microinvasion”. The accumulation of the extraductal cancer cells results in an easily measurable stellate or spherical tumour mass.

The 2nd phase of this poorly understood disease subtype is misdiagnosed by the current histopathologic terminology as ductal carcinoma *in situ* (DCIS). The use of the large format thin and thick section histopathology techniques provides sufficient visual evidence that the cancer cell accumulation in the 2nd phase is not within pre-existing ducts, but in neoducts having the hallmarks of neoductgenesis.[9,24] Basic research has provided evidence that secondary branching through epithelial-mesenchymal-transition is an invasive process which leads to new duct formation.[24,25] Management of DAB requires a re-evaluation of this misunderstood breast cancer subtype.

Conclusion

Understanding the three phases of development described here are crucial to our ability to control the aggressive subgroup of breast malignancy characterized by the fragmented casting type calcifications associated with structural dedifferentiation of neoducts. The current histopathologic view considers only two steps, “DCIS” and an invasive phase, but does not explain why the “DCIS” component can be seen in regional and distant metastases, nor why “microinvasion” may lead to breast cancer death. Likewise, the same term “DCIS” is used to describe both the 1st and 2nd phases. We need to acknowledge that the 2nd phase in the evolution of cancer originating in the major lactiferous ducts is not “*in situ*” but a duct-forming invasive carcinoma, which can undergo structural dedifferentiation. The combination of DAB and dedifferentiated DAB accounts for the high fatality of women diagnosed with this diffuse form of breast cancer.

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Conflicts of interest

The authors declare that they have no competing interests.

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