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Occult Metastatic Breast Cancer Presenting with Acute Liver Failure or Pseudo-Cirrhosis: Two Cases and Review of Literature

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1. Abstract

The development of hepatic metastases from primary breast cancer carries a poor prognosis. By imaging studies, they typically appear as mass lesions. In this report, we describe two atypical and rare manifestations of occult hepatic metastases from breast cancer through two explanatory cases and literature review. The clinical presentation of our patients was ascitic decompensation in the absence of any previous history of liver disease. In one case, CT scan showed a picture compatible with cirrhosis (pseudocirrhosis). In both cases, imaging studies were negative for secondary lesions, and trans-jugular liver biopsy represented the key move to establish diagnosis. Histological examinations demonstrated diffuse hepatic infiltration by breast cancer cells and, in the second case (pseudocirrhosis), also a severe desmoplastic reaction. These clinical presentations are representative of an unusual pattern of metastatic breast cancer, described since the 1950s, which still represents a diagnostic challenge. Indeed, literature review shows that final diagnosis was reached ante-mortem in only 22 (50%) of the 44 cases reported to date. Physicians facing patients with breast cancer and unexplained progressive liver disorders should be aware of this condition since only rapid investigation and definite diagnosis can lead to rescue chemotherapy regimens before they are precluded by liver failure.

2. Introduction

Hepatic disease associated with breast cancer is common and can be related to metastatic diffusion of the tumour or to toxic effects of systemic treatment with hormonal and chemotherapeutic agents. Hepatic metastases usually present as single or multiple lesions noted in radiological exams but, rarely, the pattern of spread can be so diffuse that they are not identified on imaging. When diffuse intrasinusoidal metastases develop, patients can rapidly progress to fulminant liver failure and develop portal hypertension [1-4]. Sometimes, this metastatic pattern can assume a cirrhosis-like appearance, which is more frequently described after chemotherapy administration [5-7]. To note, acute liver failure due to the metastatic spread of the tumour is often diagnosed post-mortem [8]. Here we present two emblematic cases in which liver failure and portal hypertension developed in patients with history of breast cancer notwithstanding radiographic exams were apparently negative for liver metastatic involvement. An overview on similar cases previously reported in literature is also provided.

3. Report of Two Cases

3.1. Case 1

A 58-year-old Caucasian woman was admitted to our hospital on June

2010 complaining of abdominal discomfort and of a progressive increase in abdominal girth associated with weight gain. She had no previous history of chronic liver disease or of alcohol abuse. Physical examination confirmed the suspicion of moderate ascites. Diagnostic paracentesis was performed, and the serum-ascites albumin gradient was in the range of portal hypertension (>1.1 g/dL). Ascites was also sent for cytology and no malignant cells were observed. Blood tests were substantially negative as well as serologic tests for hepatitis B and C. A computed tomography (CT) scan of the thorax and abdomen evidenced two hyperdense lesions in the left breast parenchyma, a marked amount of ascites and right pleural effusion. Multiple bone osteoblastic metastases were also detected. A core biopsy of the breast lump was performed, revealing a grade II invasive lobular intraepithelial neoplasia; human epidermal growth factor receptor-2 (HER2) was not overexpressed. The patient underwent three cycles of combined chemotherapy and immunotherapy with paclitaxel and bevacizumab and, subsequently, she began monthly zoledronic acid. Follow-up CT scans confirmed stable disease and showed a complete resolution of abdominal and pleural effusions, so that the patient underwent left mastectomy. After 8-months, on December 2011, blood tests showed rising tumour markers, and she was given a single dose of vinorelbine and capecitabine. The blood tests performed at the beginning of January 2012 revealed increased levels of liver enzymes, thrombocytopenia and hepatic dysfunction (increased bilirubin levels and prothrombin time). The patient newly developed ascites and ankle oedema. A repeated CT scan was negative for hepatic lesions but showed the appearance of moderate splenomegaly (Figure 1). Due to thrombocytopenia, it was opted for a trans-jugular liver biopsy, and histology was diagnostic for diffuse hepatic infiltration by a poorly differentiated carcinoma (Figure 2). On January 24th 2012, the patient died due to liver failure.

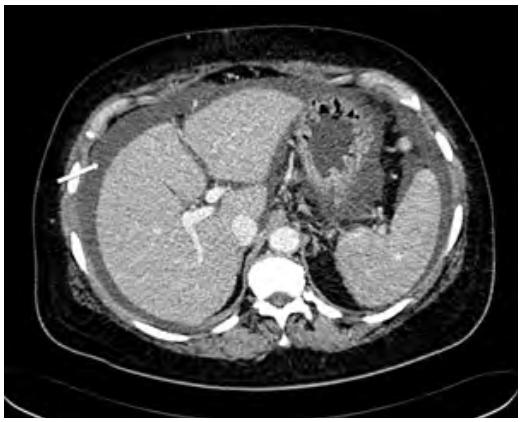

Figure 1: Computed tomography (CT) of the abdomen, case 1. CT of the abdomen (venous phase) showing the presence of ascites (arrow) and modest splenomegaly (asterisk)

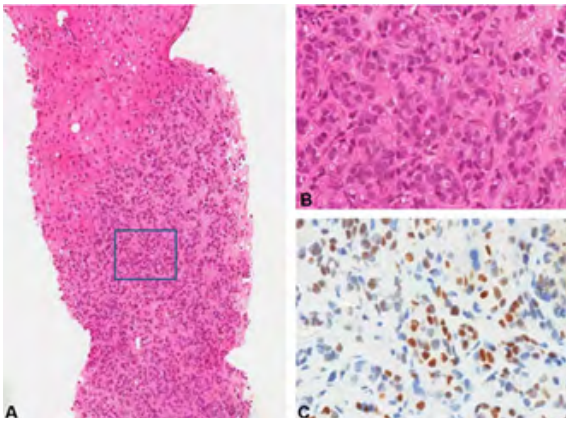

Figure 2: Liver biopsy, case 1. Panel A: hepatic metastasis from breast cancer (Hematoxylin-eosin staining, X 100); Panel B: metastatic cells with solid growth pattern (detail of panel A; Hematoxylin-eosin staining, X 400); Panel C: nuclear positivity of estrogen receptor confirms breast cancer origin (X 400).

3.2. Case 2

A 69-year-old woman was diagnosed in 1996 with a left breast multicentric lobular invasive carcinoma (grade I, T2, N0), for which she underwent left mastectomy. Three years later (in 1999), a ductal invasive plus an intraductal cribriform breast carcinoma (grade II, T1, N0) was also recognized and she underwent right quadrantectomy, followed by radiotherapy and hormone therapy, initially with tamoxifen (from 1999 to 2004), therefore with anastrozole. Starting from 2009, she continued chemotherapy with fulvestrant and she was started on zoledronic acid for the evidence of bone metastases. On March 2010, she was admitted to our unit due to jaundice, ascites and lower-extremity swelling. She had no history of chronic liver disease and she denied alcohol abuse. Serology for hepatitis viruses was negative. Liver function tests showed cholestatic hepatitis and hepato-cellular insufficiency, with level of bilirubin of 6 mg/dL and international normalized ratio of about 2. A diagnostic paracentesis was diagnostic for portal hypertension (serum-ascites albumin gradient>1.1 g/dL). Total body CT scan was negative for liver lesions and was consistent with cirrhosis and portal hypertension (lobulated hepatic margins, enlargement of caudate lobe, parenchymal dyshomogeneity) (Figure 3). The patient underwent trans-jugular liver biopsy which revealed diffuse parenchymal replacement by a poorly differentiated carcinoma and dense fibrosis (Figure 4). She developed progressive liver failure and died a few days later.

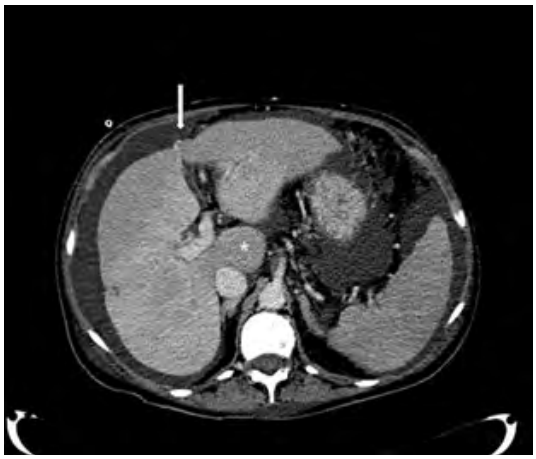

Figure 3: (CT) of the abdomen, case 2. CT of the abdomen (venous phase) showing the presence of lobulated hepatic margins (arrow) and enlargement of caudate lobe (asterisk).

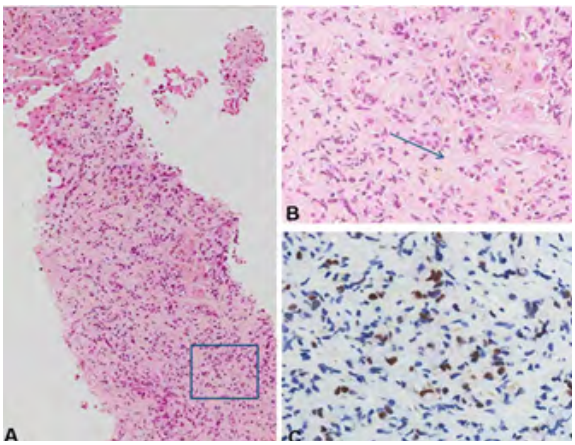

Figure 4: Liver biopsy, case 2. Panel A: hepatic metastasis from breast cancer with intense desmoplastic reaction (Hematoxylin-eosin staining, X 100); Panel B: metastatic cells with solid growth pattern and dense fibrosis (blue arrow) (detail of panel A; Hematoxylin-eosin staining, X 400); Panel C: nuclear positivity of estrogen receptor confirms breast cancer origin (X 400).

4. Discussion

Metastatic liver involvement from breast cancer is very common and carries a poor prognosis. Here we present two cases in which acute liver failure developed as a consequence of diffuse micrometastatic spread of breast carcinoma. Hepatic imaging was repeatedly negative and diagnosis was made with liver biopsy performed by the transjugular route. In the first case, the patient presented with portal hypertension due to diffuse infiltration by a poorly differentiated carcinoma. In the second case, micrometastatic spread to the liver was accompanied by a dense fibrotic reaction and a cirrhosis-like appearance (pseudo-cirrhosis). In both cases, the absence of classic discrete tumour masses on imaging studies generated a low clinical suspicion of metastatic carcinoma and a consequent diagnostic and therapeutic delay. Liver metastases usually present as hypo-dense lesions on CT images due to their hypovascular nature. However, as previously reviewed, several atypical patterns of liver metastases from breast cancer have been described [4]. These included hyper-vascular lesions, diffuse intra-sinusoidal disease, lesions causing lobar atrophy or capsular retraction and metastatic disease mimicking cirrhosis[9,10]. The mechanisms underlying diffuse parenchymal metastases remain unknown. Allison et al. proposed a role for the loss of cell surface adhesion molecule expression [8]. Indeed, in some cases of diffuse intrasinusoidal hepatic metastases, they did observe expression neither of E-cadherin nor of CD44, which are glycoproteins involved in cell-cell and cell-extracellular matrix adhesion [8,11]. The loss of E-cadherin expression is considered necessary for metastases to occur by facilitating cell detachment from the primary tumour. Moreover, in the absence of CD44, neoplastic cells may not be able to invade across endothelial cells to create large metastatic lesions. Tissue hypoxia and injury, as well as oxidative stress, could finally lead to a release of cytokines that promotes the development of liver failure in an already compromised hepatic parenchyma [8,11]. Hepatic intrasinusoidal metastatic infiltration can occur both with solid cancers of different etiology and with haematological malignancies [1-3,11-13]. Rather than creating mass lesions that are typically seen on radiological exams, cancer cells diffusely infiltrate the hepatic sinusoids and then invade branches of the hepatic and portal veins. Micrometastatic infiltration is classically described to produce a grossly enlarged liver, usually with a well-preserved shape and general architecture [8], and it is often accompanied by portal hypertension. In addition, sometimes, a strong desmoplastic reaction accompanies metastatic cell diffusion, leading to the macroscopic picture of the so-called pseudo-cirrhosis [5,14,15,16]. Although the term pseudocirrhosis has been referred also to other conditions which mimic the radiological features of liver cirrhosis [17], such as sarcoidosis or miliary metastases, it was initially used to represent the morphological changes of the liver after chemotherapeutic treatment of hepatic metastases, more frequently from breast cancer [6,7,18], but also in association with pancreatic [19], thyroid [20] and oesophageal cancers [21]. With this background, even if the pathogenesis of pseudocirrhosis is not completely clear, we should recognize and identify two different patterns of disease: 1) "toxic pseudocirrhosis", with histological features of nodular regenerative hyperplasia or of other categories of non-cirrhotic portal hypertension (NCPH), who represents the hepatic regenerative response to chemotherapy-induced vascular hepatic injury [22,23,2]; "micrometastatic pseudocirrhosis", in which, despite negative radiology, histological findings show diffuse parenchymal replacement by tumour and dense fibrosis, with an architecture resembling that of cirrhosis[14,16,24]. Finally, when hepatic intrasinusoidal metastatic infiltration occurs, independently from the specific pattern of disease observed, liver failure arises quite rapidly. Moreover, due to the negativity of imaging studies and the consequent lack of clinical recognition [8,25], the diagnosis is frequently autoptic [26].

Our literature search reveals 44 cases of occult metastatic breast cancer presenting with acute liver failure or pseudocirrhosis between 1950s and 2022 (including the two cases hereby reported) (Table 1).

A histological diagnosis of diffuse liver infiltration, without fibrosis involvement, was obtained in 59% of cases. Instead, a pseudocirrhosis-like appearance was described in 41% of cases and confirmed through histology only in 32% of cases (with a pre-mortem diagnosis in 29% of cases); indeed, histopathology samples weren't available in the remaining 9% of cases. Notwithstanding the rate of patients who received transjugular or percutaneous biopsy are increasing in the most recent literature, the post-mortem diagnosis of diffuse liver metastatic infiltration (with or without pseudocirrhosis features) remains consistent (41% of cases).

Few of the described cases presented initially with liver manifest metastatic disease, which completely disappeared after chemotherapy, and tardily with the development of pseudocirrhosis in occult metastatic disease (9%) [27-30].

The clinical course of acute liver failure was typically fulminant, with an average survival of 12 days. In literature, only four attempts with chemotherapy have been made [31-33, 29], with a response to treatment and a significant clinical benefits observed all patients.

Table 1:

First author and ref. number	Pseudocirrhosis	Diagnosis	Outcome from the admission	Notes
Micolonghi et al. (1958)[14]	No	Histology: autoptic	Death in 16 days	
Durham et al. (1961)[34]	No	Histology: autoptic	Death in less than 1 day	
Smith et al. (1961)[2]	No	Histology: autoptic	Death in 3 days	
Borja et al. (1975)[5]	Yes	Histology: autoptic	Death in 10 days	
Burnett et al. (1975) - case 1[35]	No	Histology: autoptic	Death in a few days	
Burnett et al. (1975) - case 2[35]	No	Histology: autoptic	Death in a few days	
Nouel et al. (1979)[36]	No	Histology: autoptic	Death in 3 days	
Schneider et al. (1984)[37]	No	Histology: autoptic	Death in 2 days	
Morrison et al. (1984)[38]	No	Histology: autoptic	Death in less than 1 day	
Razenberg et al. (1985)[26]	No	Histology: autoptic	Death in 14 days	
Trimble et al. (1989)[39]	No	Histology: autoptic	Death in 28 days	
Myszor et al. (1990) - case 2[40]	No	Histology: post-mortem liver biopsy	Death in 6 days	
Di Romana et al. (1993)[41]	No	Histology: autoptic	Death in 10 days	
Nieto et al. (1998)[42]	No	Histology: autoptic	Death in less than 1 day	

Martelli et al. (2000)[43]	No	Histology: autoptic	Death in 16 days	
Bégin et al (2001) - case 2[3]	No	Histology: autoptic	Death in 5 days	
Nascimento et al. (2001) - case 1[24]	Yes	Histology: transjugular liver biopsy	Death in 28 days	
Nascimento et al. (2001) - case 2[24]	Yes	Histology: percutaneous liver biopsy	Death in 28 days	
Mitchell et al. (2001)[44]	Yes	Histology: transjugular liver biopsy	Death in 21 days	
Agarwal et al. (2002)[45]	No	Histology: transjugular liver biopsy	Death in 8 days	
Lowenthal et al. (2003) - case 1[46]	No	Histology: autoptic	Death in 1 day	
Lowenthal et al. (2003) - case 2[46]	No	Histology: percutaneous liver biopsy	Death in 5 days	
Allison et al. (2004) Case 2[8]	No	Histology: percutaneous liver biopsy	Death in few days	
Nakajima et al. (2005)[27]	Yes	Histology: laparoscopic liver biopsy	Death in few days	The initial metastatic disease disappeared after chemotherapy
Sass et al. (2007)[47]	Yes	No histology: CT	Death in 1 day	
Cervoni et al. (2008)[28]	Yes	No histology: RMN	Unknown	The initial metastatic disease disappeared after chemotherapy
Fournier et al. (2010)[48]	Yes	Histology: transjugular liver biopsy	Unknown	
Graber et al. (2010)[49]	Yes	Histology: transjugular liver biopsy	Unknown	
Goswami et al. (2011)[32]	No	Histology: transjugular liver biopsy	Death in 14 days	
Simone et al. (2012) - case 1[50]	No	Histology: laparoscopic liver biopsy	Death in 14 days	
Simone et al. (2012) - case 2[50]	No	Histology: percutaneous liver biopsy	Death in 4 days	

Jüngst et al. (2013)[10]	Yes	Histology: transjugular liver biopsy	Death in 30 days	
Hanamornroongruang et al. (2013)[25]	No	Histology: autoptic	Death in 21 days	
Mogrovejo et al. (2014)[51]	No	Histology: transjugular liver biopsy	Death in 20 days	
Gulia et al. (2016)[33]	No	Histology: transjugular liver biopsy	Unknown	
This report (2016) - case 1	No	Histology: transjugular liver biopsy	Death in 30 days	
This report (2016) - case 2	Yes	Histology: transjugular liver biopsy	Death in a few days	
Adike et al. (2016) - case 6[29]	Yes	No histology: CT	Unknown	The initial metastatic disease disappeared after chemotherapy; not liver failure at the admission
Kashyap et al. (2018)[52]	Yes	No histology: CT	Unknown	
Hidalgo-Blanco et al. (2018)[53]	Yes	Histology: percutaneous liver biopsy	Death in 60 days	
Knouse et al. (2018)[54]	Yes	Histology: transjugular liver biopsy	Death in a few days	
Millard et al. (2019)[55]	Yes	Histology: percutaneous liver biopsy	Unknown	
Hoshina et al. (2021)[56]	Yes	Histology: laparoscopic liver biopsy	Death in 30 days	
Takata et al. (2022)[30]	Yes	Histology: percutaneous liver biopsy	Death in a few days	The initial metastatic disease disappeared after chemotherapy

5. Conclusion

Our work shed light on the atypical presentations of hepatic metastatic involvement from breast cancer. For any patients with unexplained liver disease during treatment, an occult hepatic involvement must be taken into account. As observed in literature, liver biopsy should be early proposed and the trans-jugular route should be considered when portal hypertension and thrombocytopenia increase the risk of bleeding. As observed in literature, liver biopsy should be early proposed [8] and the trans-jugular route should be considered when portal hypertension and thrombocytopenia increase the risk of bleeding. Indeed, only the definite diagnosis reached by liver histology can prompt to begin rescue chemotherapy regimens before they are precluded by liver failure [31].

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