

Metastatic Breast Cancer Simulating Cirrhosis with Portal Hypertension and Acute Liver Failure: A Case of “Pseudocirrhosis”

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Abstract

Case Report

Pseudocirrhosis describes morphological changes in the liver that resemble cirrhosis, but the histopathological findings are not suggestive of cirrhosis. It occurs most often in patients with metastatic breast cancer, although it has also been reported in other malignancies. As in cirrhosis, portal hypertension is often seen in patients with pseudocirrhosis. Pseudocirrhosis is a rare but important complication of metastatic cancer. We will describe the case of a 45-year-old woman with a history of breast cancer, without any previously known liver disease presenting with subacute liver failure. Initial imaging studies indicated cirrhosis of the liver without signs of focal malignancy. Full diagnostic workup was negative for specific causes of liver disease and provided no evidence of tumor recurrence. Malignant pseudocirrhosis is an unusual type of metastatic tumor, with spread representing a rare but important differential diagnosis of progressive liver failure.

Keywords: Liver failure, portal hypertension, pseudocirrhosis, breast cancer.

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INTRODUCTION

Malignant pseudocirrhosis has been defined as an uncommon pattern of metastatic spread of solid tumors and represents a rare but important differential diagnosis, although it has been described in some cases following chemotherapy for known cancers [1]. We represent the case of a patient who presented liver metastases from breast cancer simulating decompensated cirrhosis at the http stage, which thus posed a diagnostic difficulty.

PATIENT AND OBSERVATION

Patient M. E 45 years old, with a history of infiltrating ductal carcinoma of the left breast diagnosed 5 months ago but the patient did not follow up, referred to our training for abdominal distension without stopping materials or gas associated with jaundice cholestatic appearance made up of dark urine and discolored stools evolving for a month.

The clinical examination found that the patient was slowed down, disoriented in time and space, frank mucocutaneous jaundice with painless soft white lower

limb edema taking the bucket up to the ankles, the urine strip was negative, an asterixis with abdominal distension associated with sloping dullness, collateral circulation of the flanks and epigastrium, in the absence of palpable lymphadenopathy, deterioration in general condition due to asthenia, and marked but unquantified weight loss. The hemodynamic constants were correct.

A routine blood count gave the following results: hb at 11.7; insert: 183000; GB: 10550; TP was 40.5; albumin at 24, the liver test was disturbed, biological cytolysis and cholestasis ALAT at 102; AST at 98; PAL at 798; GGT at 572; BT at 141.3;BD at 130;BI at 10; the exploratory ascites puncture found a young citrine liquid poor in protein 4.9 not superinfected with a leukocyte count of 16The abdominal ultrasound showed a very abundant peritoneal effusion with a heterogeneous liver of granite echostructure, irregular contours, the hepatic arrow at 12cm, with multiple heterogeneous hypoechoic nodular lesions without dilation of the VBIH and EH. The portal trunk is dilated with a size limit of 15mm in caliber seems free, the homogeneous spleen increased in size measuring 14cm, the supra-hepatic veins are permeable.

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An abdominoplevian scan showed a heterogeneous liver of normal size, seat of two nodular formations, straddling segments VIII and VII, measuring for the largest 59*68mm, poorly limited, isodense, heterogeneously enhanced after injection of contrast product, tumor-like. The patient died a few days later, as well as the liver biopsy puncture was not done as well.

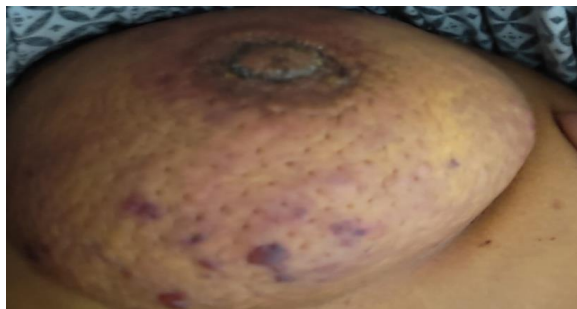


Figure 1: Appearance of the orange-peel breast



Figure 2: A heterogeneous liver of normal size (FH=12cm), seat of two nodular formations, straddling segments VIII and VII, measuring 59*68mm for the largest, poorly limited, isodense, heterogeneously enhanced after injection of contrast product, tumor-like

DISCUSSION

Pseudocirrhotic presentations have been appreciated as a model for the metastatic spread of invasive breast cancer and other malignancies [1], the clinical picture of pseudocirrhosis is represented by complications of portal hypertension and progressive liver failure.

Hepatic pseudocirrhosis is a deep peritumoral desmoplastic reaction has been reported to be mounted by diffuse neoplastic cellular infiltration, which is potentially related to altered expression, pattern of cell adhesion molecules [2, 3]. Pressure atrophy or vascular occlusion by tumor cells can lead to ischemic necrosis of hepatocytes and subsequently to acute liver failure. Indeed The pathophysiology of pseudocirrhosis is multifactorial, including liver tumor response to chemotherapy resulting in scarring and capsular retraction, fibrosis surrounding infiltrating hepatic metastatic masses, and regenerative nodular hyperplasia in response to chemotherapy ischemia induced liver damage. Regression in response to chemotherapy of

liver metastases and progression with fibrosis surrounding infiltrating liver tumors are associated with pseudocirrhosis. Sinusoidal obstruction with venous obstruction following chemotherapy is another proposed mechanism [4-6]. In contrast, the second form of "pseudocirrhosis" occurs in the absence of treated metastatic Ca, where liver histology shows signs of extensive fibrosis representing a deep desmoplastic response to the infiltrating tumor. Which shows that liver biopsy is essential to confirm the diagnosis, in our case the liver biopsy was impossible given the unstable condition of the patient [7].

Pseudocirrhosis is a radiological diagnosis describing diffuse hepatic nodularity without histopathological confirmation of cirrhosis. It is defined by morphological changes that mimic cirrhosis, including capsular retraction, nodularity, parenchymal atrophy, and caudate lobe hypertrophy [8, 6].

According to the majority of the studies carried out [9], they found that pseudocirrhosis occurs in the context of the progression of a known malignancy under systemic chemotherapy. This case illustrates an atypical presentation due to the predominance of symptoms consistent with portal hypertension and chronic liver disease, and our patient had not been exposed to chemotherapy agents, so the liver damage cannot be explained by chemotherapy. Although breast cancer with liver metastases treated with chemotherapy is the most commonly reported cause of pseudocirrhosis, it has also been reported in association with other metastatic diseases, including pancreatic cancer, colon cancer, cancer medullary thyroid and esophageal cancer [5, 10, 11].

Early recognition of pseudocirrhosis is important because it may be associated with complications of portal hypertension, rapid progression to liver failure, and increased morbidity and mortality in patients with metastatic cancer.

CONCLUSION

Determining the etiology of liver failure is crucial for the subsequent clinical management of these patients, as liver transplantation is contraindicated in patients with active malignancy.

Gastroenterologists and radiologists should remain vigilant and consider conditions that mimic with cirrhosis due to their clinical presentation and imaging findings. Early recognition may allow closer disease monitoring and focus more about symptom control.

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