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Hepar Lobatum Carcinomatosum: A Rare Complication Discovered After an Episode of Digestive Hemorrhage

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Abstract **Case Report**

Aims: Show the importance of imaging in the diagnosis and follow up of Hepar Lobatum Carcinomatosum (HLC) in the context of a breast neoplasia. ***Presentation of case***: We report a case of an episode of upper GI hemorrhage, in a 54 year old female patient, with chronic abdominal pain and asthenia. The CT scan showed a dysmorphic liver, with irregular contours, multiple capsular retractions, associated with abundant ascites. ***Discussion and Conclusion****:* HLC is defined as an acquired non-cirrhotic liver deformity secondary to liver metastases during a neoplasia, most often of the breast. The clinical presentations are not very specific and often late, and represented by signs of liver failure associated or not with portal hypertension in all its manifestations. Imaging in HLC, especially CT scan, shows similarities to a cirrhotic liver with hepatic dysmorphia, caudal lobe enlargement and segment IV atrophy. The presence of capsular retraction resulting in lobulated liver contours with reduced liver volume. It is therefore important to know the existence of this extensive form of secondary liver involvement and the set of complications that can arise.
Keywords: Hepar Lobatum Carcinomatosum, cirrhosis, Portal hypertension, CT scan**.**

INTRODUCTION

Hepar lobatum is defined as an acquired dysmorphia of the liver, which is characterized by border irregularities associated with capsular depressions. It was initially described as the final stage of tertiary syphilis [1]. The first case of hepar lobatum associated with metastatic carcinoma was described in 1924 by Busni, and was named hepar lobatum carcinomatosum [2]. This is defined as an acquired non-cirrhotic liver deformity secondary to liver metastases during a neoplasia, most often of the breast [3-5]. We report a case of hepar lobatum carcinomatosum discovered during a thoraco-abdomino-pelvic CT scan as part of the extension work-up of a late diagnosed invasive ductal carcinoma. The diagnosis in our case was suspected in front of a complication of the pathology in question.

OBSERVATION

Clinical Results

A 54-year-old female patient, not known to have a cardiovascular or digestive pathology, with a history of invasive breast carcinoma treated by surgery and chemotherapy for which she was no longer being followed. She presented to the emergency room with a first episode of upper GI hemorrhage of sudden onset. On admission, the patient was conscious, in good general condition, with a blood pressure of 110/70mmHg, respiratory rate of 22cpm, and heart rate of 80bpm. On questioning, the patient reported chronic abdominal pain for more than six months, in a context of asthenia. A careful clinical examination revealed signs of chronic liver disease including abdominal distension, subicterus, hepatomegaly, cutaneous stellate angiomas and palmar erythrosis.

Biological Results

In the context of her chronic liver disease, a biological check-up was requested, showing thrombocytopenia, a hemoglobin level of 12, a high bilirubin level (45umol/L), and a low protein V level of 30.

There were no other clinical signs pointing to syphilis, especially no sexual activity at risk. She had no history of drug or alcohol use. Therefore, no further tests were requested.

Imaging Results

In the context of the patient's known and documented history of breast carcinoma, a thoraco-abdomino-pelvic CT scan was requested to evaluate the status of her liver, the status of her neoplasia and possible secondary localization. The CT scan showed a dysmorphic liver, with irregular contours, multiple capsular retractions, heterodense, associated with abundant ascites (Figure 1). The patient had already undergone a previous CT scan, performed one year earlier, which showed secondary liver damage, including a normal-sized liver with bumpy contours and confluent nodules in places, without capsular retraction or hepatic dysmorphism, associated with mixed axial and peripheral skeletal damage.

As part of the exploration of his upper digestive hemorrhage, an oesophageal fibroscopy was requested, showing stage I oesophageal varices.

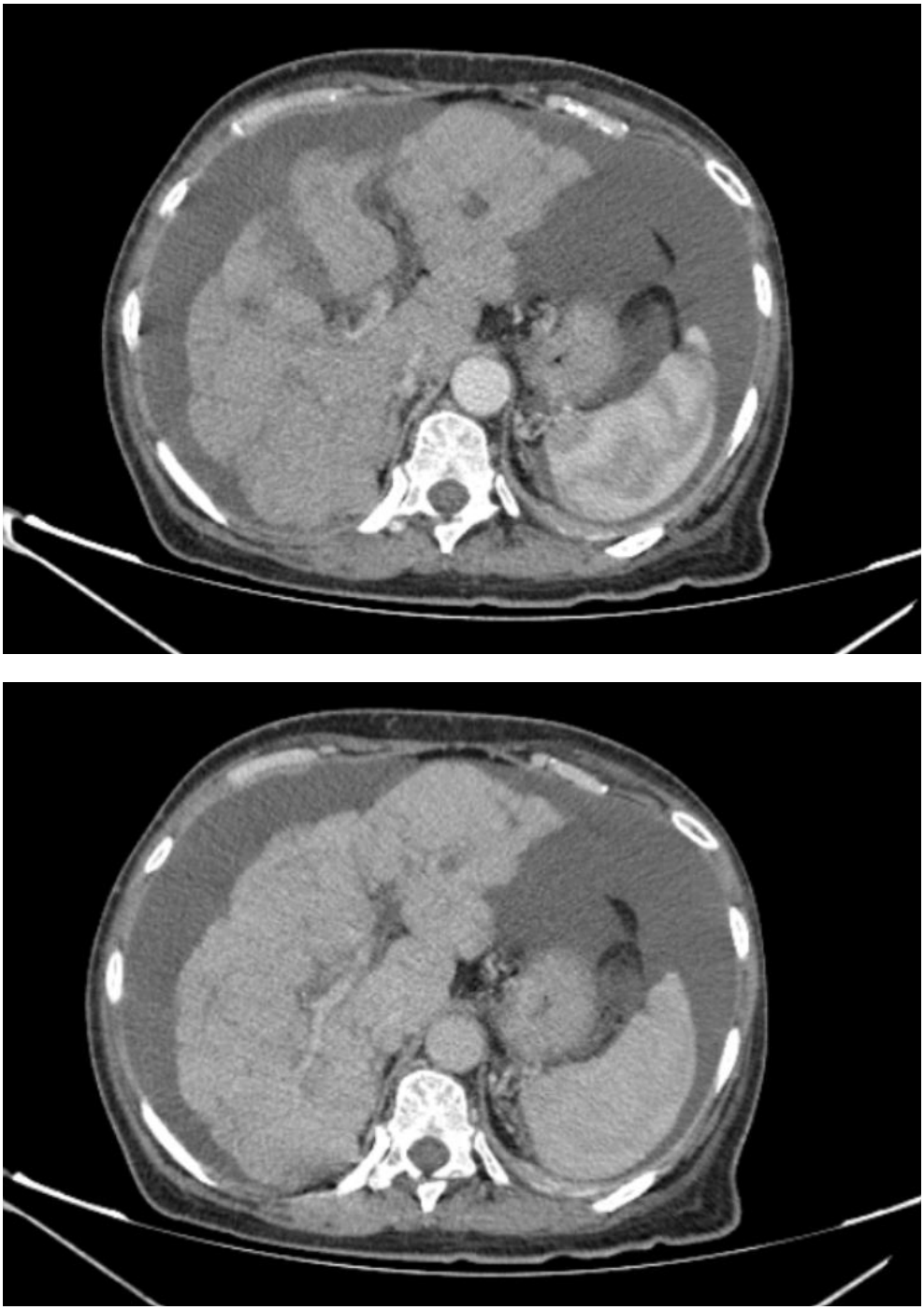

Figure 1: Abdominal CT, axial section (injection at portal phase): dysmorphic liver with multiple deep contour changes, related to capsular retractions, giving a hepar lobatum appearance. It is associated with abundant ascites

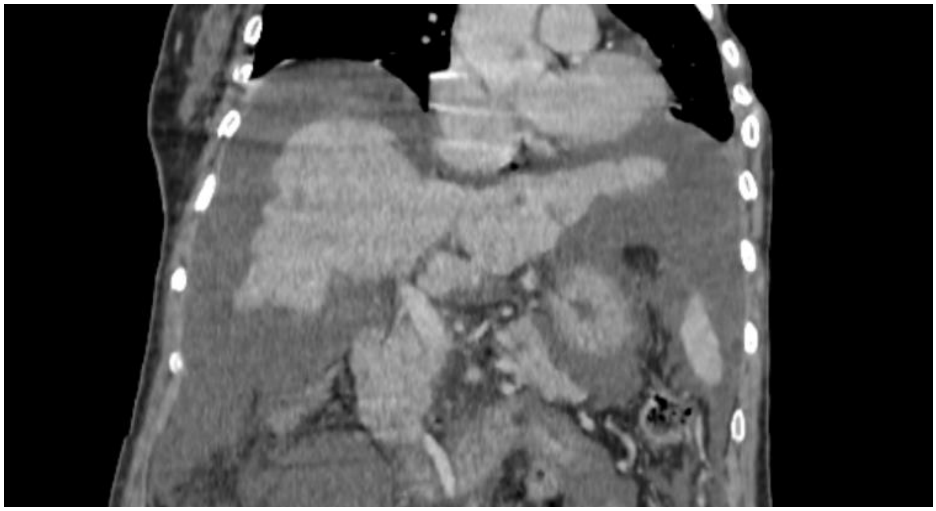

Figure 2: Abdominal CT scan, coronal section of the same scan performed in our patient; objectifying the aspect of hepar lobatum cariconomatosum (injection at portal phase)

Pathological results: No percutaneous biopsy was performed in this patient.

DISCUSSION

In this report, we will discuss liver impairment at the stage of hepar lobatum carcinomatosum (HLC) secondary to liver metastases in the context of invasive metastatic breast carcinoma treated by a combination of surgery and chemotherapy.

This diagnosis was based on a number of arguments, in particular the presence of a previously known and treated invasive ductal breast carcinoma, even if lost to follow-up, the presence of portal hypertension, and finally the imaging after an accurate and adequate reading of an abdominal CT scan showing liver dysmorphia associated with capsular fibrous retraction at the level of the initially metastatic sites.

The diagnosis of cirrhosis was ruled out due to the absence of risk factors that could lead to chronic hepatopathy, in particular no consumption of alcohol or hepatotoxic herbs, no history of viral hepatitis.

HLC, also called pseudocirrhotic liver [1], is the rarest form of liver metastasis, few cases have been reported in the literature, the first case of which was discovered by Busni in 1924, on an autopsy report of a 37-year-old woman being followed for carcinoma of the breast, where he described a specific form of liver metastasis [6].

HLC is already described in association with breast cancer, however initially described during tertiary syphilis as a result of syphilitic gum scarring of the liver, more and more cases have been reported in the setting of breast neoplasia as an advanced stage of secondary liver involvement. HLC is often associated with invasive ductal or lobular breast carcinoma, however, other cases of liver metastases in the setting of kidney, stomach, or nasopharyngeal cancer have been associated with this involvement, and in rare cases HLC was revealing of the underlying cancer [6].

Its pathogenesis, said to be multifactorial, is not yet completely elucidated, however, hypotheses have been established: it is characterized by the combination of cancerous scars associated with compensatory hyperplasia of the healthy hepatic parenchyma. At least two factors are considered to be responsible for carcinomatous liver scars: a stromal reaction to the carcinoma or a vascular abnormality secondary to infiltration such as occlusion of the portal trunk, the hepatic veins or their branches. Hypoperfusion will be responsible for a heterogeneous liver with atrophy of the affected segments and hypertrophy of the healthy parenchyma [7-9].

The stromal desmoplastic reaction is an excess production of fibrous tissue by activation of each of the cellular components of the stroma. It could result from the production by tumor cells of myofibroblastic growth factors such as PDGF (platelets deriverd growth factor), fibroblast growth factor-2, and transforming growth factor, leading to an increase in extracellular matrix synthesis.

In some cases, chemotherapy has been reported to be responsible for the development of HLC. The hepatotoxic effect of chemotherapy ranges from elevated liver enzymes in the blood to fibrosis, portal hypertension and cirrhosis. Several studies have attempted to demonstrate a direct link between chemotherapy and secondary liver cirrhosis, the most commonly incriminated drugs being methotrexate and 6-mercaptopurine according to a study by Shikhoda and Baird investigating morphological changes in the liver on CT imaging in patients followed for metastatic breast carcinoma undergoing systemic chemotherapy and chemotherapy directly injected into the liver artery. The result of this study was hepatic steatosis, cirrhosis,

localized atrophy and areas of hepatic hypodensity, suggesting that the hepatic morphological variations were secondary to treatment, however its study was limited by the lack of histological evidence. In another prospective study in patients with different benign and malignant liver diseases, capsular shrinkage adjacent to liver tumors was specific to malignant tumors, which allows the HLC to be related to secondary metastases rather than to treatment-related hepatotoxicity [10].

The clinical presentations are not very specific and often late, and represented by signs of liver failure associated or not with portal hypertension in all its manifestations. The association of this picture with a breast carcinoma, and a confirmation by imaging can spare the realization of invasive biopsies to confirm the diagnosis, this in the absence of anamnestic or clinical criteria directing towards another origin of the hepatocellular insufficiency.

Imaging in HLC shows similarities to a cirrhotic liver with hepatic dysmorphia, caudal lobe enlargement and segment IV atrophy. The presence of capsular retraction resulting in lobulated liver contours with reduced liver volume. In patients with focal or isolated metastases, parenchymal changes may be limited to these sites, with atrophy of the affected liver lobe, which explains the interest of a good CT follow-up with comparison of the old imaging. Nodules of regeneration similar to those of cirrhosis may be associated, but localized to the sites of metastases already documented if available. This was the case in our patient with the presence of liver dysmorphia in its complete picture, capsular retraction, the presence of ascites as well as clinical and CT signs of portal hypertension.

CONCLUSION

Hepar lobatum carcinomatosum remains a rather rare form of advanced liver metastases of breast carcinoma, it is important to know the existence of this extensive form of secondary liver involvement and the set of complications that can arise. The CT appearance is quite typical and a comparison with previous imaging is necessary to link the liver dysmorphia to metastases. However, other causes of cirrhotic liver damage must be ruled out.

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