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Chest wall metastasis from hepatocellular carcinoma in the absence of a primary: An unusual presentation

ABSTRACT
Metastatic hepatocellular carcinoma (HCC) has an aggressive course, with a very poor outcome. The common hematogenous metastatic sites are the lungs, bones, and adrenal glands. The chest wall is an extremely rare site of metastasis from HCC. We report a rare presentation in a gentleman, where the chest wall metastasis kept progressing in spite of treatment, without any evidence of a detectable primary.

KEY WORDS: Chest wall, hepatocellular carcinoma, metastasis

Metastatic hepatocellular carcinoma (HCC) has an aggressive course, with a very poor outcome manifesting within a few months.[1,2] Among all the metastatic sites, bone metastases is the commonest, while chest wall metastases are extremely rare.[3-5] We report a rare presentation in a gentleman where, without any evidence of a detectable primary, a chest wall metastases kept progressing in spite of treatment.

CASE REPORT
A 65-year-old gentleman presented to our hospital with a history of a painful swelling on the left chest of three months’ duration.

Prior to being seen in our hospital he had been evaluated at a local hospital around 8 months earlier. A CT scan of the thorax and abdomen done at that hospital had shown a large soft tissue mass in the left thoracic region, associated with a destructive lesion of the left 5th rib; the mass had shown a large extrapleural component, with displacement of the pleura. There was no other disease seen elsewhere. He underwent excision of the tumor and the histopathology review at our hospital showed that the tissue was consistent with HCC, while the presence of vascular emboli suggested that it was a metastasis from a HCC. He had received six cycles of adriamycin-based chemotherapy elsewhere. The tumor was found to be infiltrating the surrounding muscle.

After he presented at our hospital, a follow-up CT scan showed an increase in the size of the mass in the left thorax, with extensive multiple rounded nodular scattered opacities in both the lung fields, representing secondary metastases in the lung parenchyma. There was no lesion seen in the liver, which was also seen to be noncirrhotic. Serum alpha-fetoproteins were marginally elevated. There was no evidence of hepatitis and all the viral markers were negative. The blood counts and biochemistry were normal.

In view of his poor general condition and the progressive disease, he was offered palliative and best supportive care. The patient died at home, 3 months after being last seen in our hospital, with progressive metastatic disease being the most likely cause.

DISCUSSION
HCC has an aggressive course. The 5-year survival in patients with symptomatic HCC is 0.8% in men and 4.4% in women after the onset of symptoms.[4]

The gene expression profiles of liver parenchyma among HCC patients may be different. Budhu et al. have stated that many of the metastasis-promoting genes are embedded in the primary tumors and that the ability to metastasize may be an inherent quality of the tumor from the beginning.[5] The condition of the liver parenchyma, the degree of viral hepatitis-mediated liver damage, or the genetic makeup of individuals may have a significant role to play in the development of metastases.[5]

Hematogenous metastases is common, and the usual sites are lung (49%), bone (16%), adrenal glands (15%), pancreas (4%), kidney (3%), and the
spleen (2%), as ascertained in an autopsy series. Katyal et al. reported a high rate of lymphatic spread in HCC, which was seen in 78 (53%) of 148 patients with extrahepatic HCC. Malignant chest wall masses arising from a primary focus in the liver are rare. In the presence of a normal liver, deposits of HCC in the chest wall (whether isolated or multiple) is exceptional.

Ectopic liver is a rare developmental error where liver tissue rests are found at sites such as the gall bladder, hepatic ligaments, omentum, retroperitoneum, and the thorax. A survey of literature found 20 reports of cases of ectopic liver that developed HCC, while the mother liver did not have HCC. An extrahepatic HCC without a primary intrahepatic lesion has been explained as being either due to ectopic liver carcinogenesis or hepatoid adenocarcinoma.

Hepatoid adenocarcinoma is a variant form of adenocarcinoma, characterized by vast hepatic differentiation. It produces alpha-fetoprotein, while having the same function and form as HCC. It originates from the endodermal mucosa and therefore can manifest itself in the gastrointestinal tract, ovaries, pancreas, lungs, kidneys, uterus, or bladder.

In our patient, the possibility of an extrahepatic HCC was considered, but in view of the presence of vascular emboli on histopathology and the development of multiple lung metastases, the diagnosis of metastatic HCC was favoured.

Surgical excision and infusional chemotherapy in localized metastases have been shown to provide reasonable control. It has been suggested that those with poor performance status and poor hepatic reserve should receive only supportive care, while in those with good performance status and hepatic reserve, anticancer therapy may be administered. The outcome is usually dismal in such patients.

To summarize, the presentation in this patient was unique because of progression of the metastases in spite of the absence of a primary. There are no guidelines available regarding anticancer therapy in patients with extrahepatic spread from a HCC. The aim should be to achieve good palliation in such patients.

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