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Case Report Open Access

A Rare Case Report of a 79-years Old Male with Hepatoid Adenocarcinoma of the Lung

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Abstract

Hepatoid Adenocarcinoma of Lung Cancer (HAL) is a very rare form defined as an Alpha-Fetoprotein (AFP)-producing primary lung carcinoma with specific morphological features resembling hepatocellular carcinoma. Pathological diagnosis was reported as Non-Small Cell Lung Cancer (NSCLC), compatible with hepatoid carcinoma or AFP-producing tumor.

Keywords: Hepatic Adenocarcinoma; Lung; Non-Small Cell Lung Cancer

Abbreviations: HAL: Hepatoid Adenocarcinoma of Lung Cancer; AFP: Alpha-Fetoprotein; RLL: Right Lower Lobe; IHC: Immunohisto chemical Staining; PD: Progressive Disease; NSCLC: Non-Small Cell Lung Cancer; CT: Computed Tomography; PD: Progressive Disease

Introduction

Hepatoid tumor or hepatoid (adeno) carcinoma are terms for a number of uncommon or rare neoplasms in humans, named for a visual resemblance of the cells under the microscope to those of hepatocellular carcinoma, the most common form of liver cancer. They can arise in several parts of the body, and thus form sub-types of diseases such as stomach cancer and pancreatic cancer.

Adenocarcinoma of the lung is the most common type of lung cancer, and like other forms of lung cancer, it is characterized by distinct cellular and molecular features. It is classified as one of several non-small cell lung cancers (NSCLC), to distinguish it from small cell lung cancer which has a different behavior and prognosis. Lung adenocarcinoma is further classified into several subtypes and variants. The signs and symptoms of this specific type of lung cancer are similar to other forms of lung cancer, and patients most commonly complain of persistent cough and shortness of breath.

The pathophysiology of adenocarcinoma is complicated, but generally follows a histologic progression from cells found in healthy lungs to distinctly dysmorphic, or irregular cells. There are several distinct molecular and genetic pathways that contribute to this progression. Like many lung cancers, adenocarcinoma of the lung is often advanced by the time of diagnosis. Once a lesion or tumor is identified with various imaging modalities, such as computed tomography (CT) or X-ray, a biopsy is required to confirm the diagnosis.

Treatment of this lung cancer is based upon the specific subtype and the extent of spread from the primary tumor. Surgical resection, chemotherapy, radiotherapy, targeted therapy and immunotherapy are used in attempt to eradicate the cancerous cells based upon these factors.

Case Presentation

This contextual investigation of this case study chiefly addresses the 79-year-old male patient who was suffering from chronic cough and weight loss from past three months. The patient had two months of chemo-holiday then he developed Progressive Disease (PD). Pathological diagnosis was reported as Non-Small Cell Lung Cancer (NSCLC), compatible with hepatoid carcinoma or AFP-producing tumor. Unfortunately, the patient didn't react to Pemetrexed with expanding of right pleural emanation and growth of right hilar mass including deteriorating of manifestations. The patient died following 3 weeks of oral etoposide because of no reactions of treatment.

A 79-year-old male presented with chronic cough and weight loss in 3 months. He had a history of 20 pack-year smoking and stopped for 2 months. Chest x-ray at baseline showed huge right hilar mass which was concordant with chest CT demonstrating lobulated mass size 8.8 × 8.8 × 10.7 cm at Right Lower Lobe (RLL), enlargement of right hilar and sub-carina lymph nodes without abnormal liver lesion. Bronchoscopy with tissue biopsy at RLL was performed.

Chest x-ray at standard showed colossal right hilar mass which was concordant with chest CT exhibiting lobulated mass size 8.8 × 8.8 × 10.7 cm at Right Lower Lobe (RLL), amplification of right hilar and sub-carina lymph nodes without unusual liver sore. Bronchoscopy with tissue biopsy at RLL was performed. Pathological diagnosis was reported as Non-Small Cell Lung Cancer (NSCLC), compatible with hepatoid carcinoma or AFP-producing tumor. Immunohistochemical (IHC) staining showed strongly positive of AFP, hepatocyte, AE1/AE3, and focally positive of CD117 while PLAP, CD30, synaptophysin and chromogranin A were negative.

Results

Immunohistochemical (IHC) staining showed strongly positive of AFP, hepatocyte, AE1/AE3, and focally positive of CD117 while PLAP, CD30, synaptophysin and chromogranin A were negative in Figure 1.

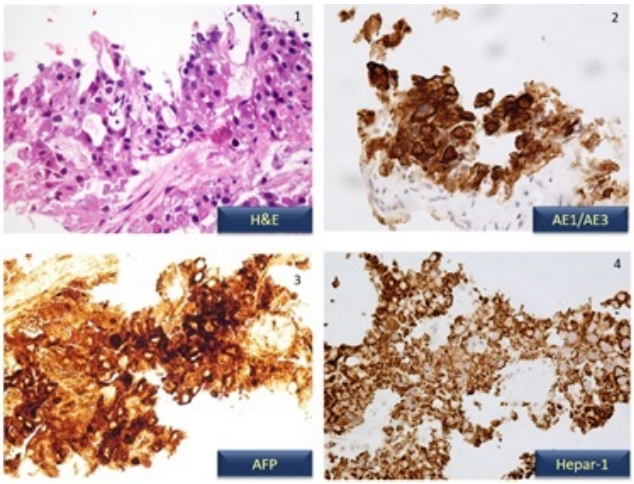

Figure 1: 1: H&E slide show adenocarcinoma of lung tissue with distinct cell borders and abnormal nuclear chromatin including grandular characteristics. 2: AE1/AE3 straining pattern. 3: AFP staining pattern. 4: Hepar-1 staining pattern.

AFP at baseline was 60,500 ng/mL. The staging was T4N2M0 (stage IIIB). The initial treatment was palliative chemotherapy with Carboplatin/Etoposide due to poor performance status (PS; ECOG=2) and emphysematous lung disease. Dramatic response in symptoms was observed after 3 cycles of chemotherapy with the tumor shrinkage more than 50% in Figure 2.

Figure 2: Significantly tumor response after palliative chemotheraphy with carboplatin/etoposide at C3 cycle.

AFP was gradually decreased to 8,824 ng/mL after cycle number 6. The patient had 2 months of chemo-holiday then he developed Progressive Disease (PD) and AFP was increasing to 32517 ng/mL shown in Figure 3. Single agent Pemetrexed was initiated due to poor PS. Unfortunately, the patient did not respond to Pemetrexed with increasing of right pleural effusion and enlargement of right hilar mass including worsening of symptoms. Prognosis, options of treatment, and palliative care were discussed with patient and family. Finally, the patient chose to try oral Etoposide. Again, there was not any responses. The patient passed away after 3 weeks of oral Etoposide.

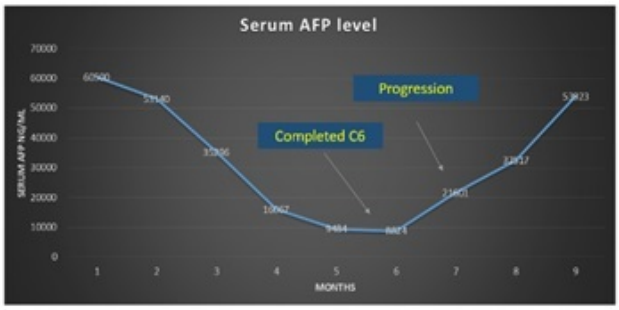

Figure 3: AFP level after the treatment with chemotherapy was gradually decreased.

Discussion

First report of HAL was described by Ishikura defined as an AFP-producing carcinoma containing a mixture of tubular and papillary adenocarcinoma with sheet-like or trabecular proliferation of neoplastic cell, contain eosinophilic cytoplasm and centrally located nuclei that resembling HCC cells [1]. These morphologies were present in this case with elevate AFP level that confirm diagnosis of AFP-producing tumor or HCC. Distinguishing HAL form lung metastasis from HCC requires clinical, radiologic, morphologic feature and IHC correlation. IHC staining for AFP and Hepar-1 help to diagnosed neoplastic Hepatocytes. Previous reports found AFP expression in all studies; However, hepar-1 expression is positive in only one study that also resemble to this patient [2].

AFP level was elevated in previous studies [3]. This result may be tumor marker in treatment response and follow up patient. There is no standard treatment and prognosis because of the small number of cases that have been described and followed up. However, treatment should depend on staging and multimodality treatment. There are one case report presents sorafenib in combination with platinum-based chemotherapy, which led to partial response [4,5].

Conclusion

There are 16 case reports of hepatoid adenocarcinoma of lung in the world literature. We have reported new case of HAL. Due to patient's age, poor performance status and advance stage in this patient, platinum doublet is popper management and dramatic response to treatment with 9 months of survival.

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