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General Medicine

CASE REPORT OF 60 YEAR OLD MALE WITH HEPATOCELLULAR CARCINOMA

KEY WORDS:

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ABSTRACT

Around 90% of liver cancer cases are hepatocellular carcinoma (HCC), making it the most frequent kind of liver cancer. At present, the need for molecular information necessitating tissue or liquid samples poses a difficulty to non-invasive criterion-based diagnosis. Here we discuss the case of a 60-year-old guy who has a very probable diagnosis of hepatocellular carcinoma.

INTRODUCTION

About 80% of all occurrences of liver cancer are hepatocellular carcinoma (HCC), which is formed from hepatocytes and is the main type of liver cancer. The tumor's stage and the presence of preexisting cirrhosis determine how hepatocellular carcinoma (HCC) manifests.[1]

- In its early stages, non-cirrhotic linked HCC may not cause any symptoms. When HCC first manifests clinically, the average patient is 69 years old.
- HCC patients with chronic hepatitis C symptoms of quickly worsening liver failure include: delays, itching, confusion, ascites, palpable mass in the upper abdomen, fever, exhaustion, fast satiety, abdominal protrusion, weight loss, early satiety, abdominal distention, cachexia, and feverishness. One of the most prevalent symptoms of HCC is discomfort in the abdomen.[2]
- Symptoms of the paraneoplastic syndrome in patients with head and neck cancer (HCC) include low blood sugar, red blood cell count, high calcium levels, diarrhea, and skin abnormalities include pemphigus foliaceous, pityriasis rotunda, dermatomyositis, and the Leser-Trelat sign.[3]

Hepatic encephalopathy, pyogenic liver abscess, obstructive jaundice, intra-abdominal bleeding and variceal bleeding are among the many conditions patients may suffer from.

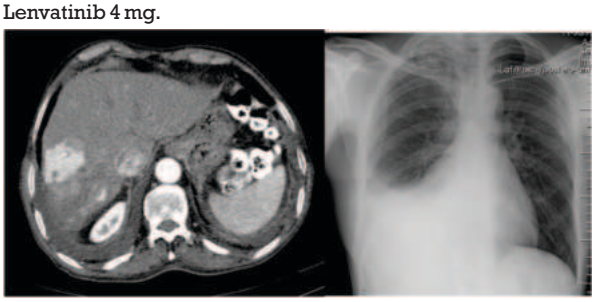
Environmental and genetic causes help in building the root of HCCs. The cirrhosis determines the most common etiology of HCC which takes place in approximately 80 % of those suffering. Nevertheless, any chronic inflammatory disease of the liver can turn the disease.Nearly all of these tumors (90–95%) develop as a biological response to chronic hepatitis B and C virus (HBV and HCV, respectively).[4]

Hepatitis B virus (HBV) oncogenesis mostly occurs as a result of genome integration with the host genome. Disorders such as iron overload cirrhosis (hemochromatosis), persistent alcoholic cirrhosis, alpha-1 antitrypsin deficiency, tyrosinemia, and cirulous hepatitis B and C are among the diseases that increase the risk of hepatocellular carcinoma (HCC). [5] Alcohol can precipitate HCC via cirrhosis, a secondary way in which drinking could impact HCC. HCC patients prognosis depends on a stage of their disease at the time of its diagnosis. With an earlier diagnosis and a corresponding treatment, on should be able to expect a five-year survival rate, even if one's condition advances and progresses at a faster rate. The greatest age-specific incidence was recorded in adults older than 70 years of age, indicating that aging is a significant risk factor. Additionally, HCC is more common in males.[6]

Case Presentation

60-year-old male decorator by occupation came to the OPD with Complaints of Breathlessness since 3 days, abdominal distension since 15 days, Cough with sputum expectoration since 15days and altered sleep pattern since 10 days. Patient was apparently normal 15 days back after which he had developed abdominal distension, which was progressive in nature, then He developed cough with expectoration since 10 days back. From Last 3 days the patient started developing Breathlessness, which was progressing from MMRC Grade 2 —> 3. Patient is Known case of DCLD for last 3 years with HBsAg Positive. Known Case of Type 2 Diabetes Mellitus and on Medication. Anorexia present. Sleep pattern irregular. On Examination Patient is Conscious, oriented, afebrile, Moderately Built and Moderately Nourished. Gynaecomastia Present on Both the Sides. Patient vitals at the time of admission was stable. There were No Signs of Pallor skin,

icterus, clubbing, cyanosis, or lymphadenopathy. On Local Examination abdomen was distended, flanks were full on both sides, Fluid Thrill Present, Non-tender. Patient was evaluated with necessary investigations. Total Bilirubin - 2.0, Direct Bilirubin - 1.2, Total Protein - 5.5, Albumin - 2.2, Globulin - 3.3. INR - 1.78. Chest Xray PA view shows Right Sided Pleural Effusion. USG abdomen shows 1) Parenchyma Disease; Calculus Cholecystitis; Splenomegaly; free floating echogenic content in perihepatic, Morrisons pouch, bilateral paracolic gutter and pelvic region likely to be haemorrhagic. CECT Abdomen shows: Cirrhotic morphology of the liver with multiple ill-defined hypodense lesions in II/IVB, VI, VII & VIII segments showing heterogeneous and subcapsular enhancement - Features likely suggestive of hepatocellular carcinoma. * Splenomegaly. * Ascites. Diagnostic and therapeutic Ascitic Tapping was Done and around 1.8 Liter of haemorrhagic fluid was tapped, following ascitic fluid results shows Protein 1.1g/dl, Albumin -0.4g/dl (SAAG - >1.1g/dl, Transudative fluid). Serum AFP -527 ng/ml. Pulmonologist Opinion was obtained in view of persistent Breathlessness even after ascitic tapping, and Thoracocentesis was done in view of pleural effusion, around 600 ml of haemorrhagic fluid was tapped. Opinion was Obtained from Hepatologist and Opined as HBV related Cirrhosis and Advised to Start On Lenvatinib 4 mg.



DISCUSSION

Hepatocellular carcinoma, as demonstrated above presented with Cirrhosis of liver secondary to chronic hepatitis B infection.[7] On the first assessment, it is possible to see high levels of liver function tests such as albumin, bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), etc.[8] This might be a sign of how bad the illness is. Thrombocytopenia, anemia, hyponatremia, hypoglycemia, an increased international normalized ratio (INR), prothrombin time (PT), and impaired synthetic liver function or reserve are further aberrant laboratory results. Patients exhibiting these symptoms are most often seen in those with severe HCC, chronic hepatitis, or HCC caused by cirrhosis.[9] The presence of normal serum enzymes does not necessarily mean that patients with early non-cirrhotic-related HCC had them detected for the first time. Patients exhibiting paraneoplastic characteristics of HCC may exhibit hypoglycemia, hypercalcemia, and erythrocytosis.[10]

Various other laboratory tests may be used to determine the cause of HCC, including as those for hepatitis B surface antigen, anti-HCV antibodies, levels of copper and iron saturation, and alpha antitrypsin.[11]

Ultrasound works well for identifying HCC tumors bigger than 2 cm, but it doesn't do a very good job at detecting lesions smaller than that. Therefore, computed tomography is now the gold standard for detecting HCC, rather than ultrasound.[12]

There are three stages of contrast tomography in HCC: the arterial phase (which takes 2–40 seconds after injection) and the portal vein phase that (occur 50–90 seconds after injection), at which the liver parenchyma is clearly seen as a result of this injection. Due to its identification of regenerating nodules with the early high-fat HCC enabled by T1-seqnecing, MRI is believed to play a role in image diagnosis process of HCC.[13]

The 591 amino acid glycoprotein known as alpha fetaprotein (AFP) gets significantly expressed in HCC patients. Elevated AFP levels are seen in several gastrointestinal tumors as well as benign liver disorders such as cirrhosis and hepatitis. After HCC is entirely removed, AFP levels usually go back to normal rather quickly. Due to the persistence of low AFP-producing metastases, such low AFP levels do not rule out recurrence.[14]

The software for the diagnosis of HCC The definitional criteria should be established which will be pathologicaly used for the nodules less than 2 cm in size. In tiny HCC less than 2 cm in diameter, the accuracy of a fine-needle biopsy might vary from 50% to 70%, depending on the size of the lesion. With the existing diagnostic methods, it is very hard to accurately detect HCC from nodules smaller than 1 cm, and only 50% of them will be HCC. Histopathologists have a hurdle when trying to distinguish between preneoplastic lesions and early, well-differentiated HCC. if a nodule is greater than 2 cm in size in a patient with liver cirrhosis, positivity in one more imaging technique correlating with AFP levels higher than 400 ng/ml, or the simultaneous results of two types of imaging studies (ultrasonography, spiral-CT or MRI) revealing arterial hyper vascularization would result in a definitive HCC diagnosis.

To stage the disease, BCLC stages system, is mostly used for HCC. The safeguards look at three key thresholds: tumor status (number, size, vascular invasion, extrahepatic localization), liver function (Child-Pugh score) and performance status (PS, based on the Eastern Cooperative Oncology Group scale6). These limits are used together to categorize patients into five epidemic stages (0, A, B, C, and D), and treatment option are either When it comes to objectivity, the third defining feature of variables, which includes rhetoric, postulates the least among the three types. If the patient's performance status (PS) is perfect (PS 0) where the liver function also remains healthy (Child-Pugh A) and the tumor is considered to be less than 2 cm in size without any signs of invasion to the veins or metastasis it is referred to as an early stage (BCLC 0).[15]

The BCLC differentiation is into two stages. Patients with metastatic disease or other noticeable symptoms are considered of advanced stages. The patients with a single nodule less than 5cm or less than 3cm up to 3 nodules or less than 3cm are considered to belong in stage I of the BCLC. Concurring with the BCLC guidelines, surgical removal of the liver tumor otherwise referred to as resection, liver transplantation, or local ablation serves as curative options to the patients in stage0 and A. Patients without symptoms in stage BCLCB have big or multifocal tumors confined to the liver parenchyma.

Those patients who show up in the clinic with symptoms which were caused by the underlying hidden cancer, by the macrovascular involvement, or by the extrahepatic dissemination of the disease are defined as being in the advanced stage (BCLC C). Another line of approach is systemic sorafenib as well as TACE for patients with BCLC B & C. For these patients who have already passed the stage where their poor PS or liver function (Child-Pugh C) suggest severe tumor or damage due to cirrhosis (BCLC C) the only therapeutic option is supportive care.

A definitive advantage of preoperative chemoembolization has not been shown. By stimulating the development of the unaffected lobes, preoperative embolization of the afflicted lobes' hepatic arteries and portal veins likely has good effects. Yet, there is a chance that ischemia may encourage tumor development and angiogenesis in malignant hepatocytes as a result of this operation.56 For some

circumstances, ethanol ablation or thermos-ablation might serve as a transitional step before undergoing surgical excision or transplanting.

CONCLUSIONS

Patients in their latter years are more likely to be affected by HCC, a kind of malignancy. Because it often progresses asymptomatically and most cases are well advanced with a poor survival probability when symptoms appear, identification is challenging. In order to lower death rates and start therapy sooner rather than later, early diagnosis is crucial.

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