Hepatocellular Carcinoma Classification and Current Treatment

Robert Elliott

*Corresponding author

Robert Elliott
Department of Imaging Sciences, University of Rochester,
601 Elmwood Avenue, Rochester, NY, 14642, United States

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Abstract

Hepatic cirrhosis is a risk factor for the development of the aggressive primary liver cancer known as hepatocellular carcinoma (HCC). The Hepatitis C virus, which causes cirrhosis, is becoming more common in the United States, which has led to an increase in HCC cases. When tumours are modest, curative treatment for HCC is indicated and may entail radiofrequency ablation, liver transplantation, or surgical resection. Transarterial chemoembolization and transarterial radioembolization are two locoregional treatments that can be utilised either as a primary therapy or as an adjuvant to surgical care. The initial monitoring of individuals at risk for HCC, the most recent recommendations for HCC diagnosis and staging, and a review of the best practises for HCC therapy will all be covered in this review article.

Keywords

Hepatocellular carcinoma, Locoregional therapy, Radiofrequency ablation, Transarterial chemoembolization, Transarterial radioembolization

Introduction

The most frequent primary liver tumour and the leading cause of death for those with cirrhosis is hepatocellular carcinoma (HCC). [1] HCC is some of the poorest survival rates of all malignancies, with 1-year and 5-year survival rates of 50% and 20%, respectively. [2] Since 1980, the incidence of HCC has tripled in the United States, mostly due to an increase in the frequency of hepatitis C among baby boomers. Population. Due to the rising incidence of cirrhosis attributable to non-alcoholic fatty liver disease (NAFLD) in the United States, it is expected that the incidence of HCC will continue to be high in the future. [3] Transarterial chemoembolization (TACE) and transarterial radioembolization with yttrium-90 (Y90) will be recommended for many patients with HCC as part of a multimodal strategy to either downstage illness (to a more manageable stage) or to treat advanced disease (to a more severe stage), a point where a transplant or surgical resection may be necessary, stop disease development (especially for those on a Waitlists for liver transplants, so-called “bridge” therapies, or more generally, palliative care with the goal of extending survival for patients who are not candidates for surgery. The current best practises for initial HCC surveillance, diagnosis, disease staging, and treatment are reviewed here.

HCC SURVEILLANCE:

Surgery is typically avoided once tumours are multifocal, above 5 cm, or show vascular invasion because at that moment undetected extrahepatic dissemination is more likely and is linked to an early disease recurrence. However, surgical indications remain debatable. Only 30–40% of individuals who are diagnosed with advanced HCC are therefore candidates for surgery. [4] Patients have a 70% likelihood of receiving an early or very early diagnosis if they are successfully enrolled in a surveillance programme. Early diagnosis is crucial in these situations since curative therapies provide 5-year survival rates that consistently exceed. According to major US guidelines, HCC surveillance is recommended for anyone with cirrhosis, regardless of the cause. [6, 7] A subpopulation of people with hepatitis B may exist who who do not yet have cirrhosis but are at such a high risk of acquiring HCC due to either high viral numbers or an Asian or African ancestry that surveillance is financially advantageous. [8] In a similar vein, although this is not known for sure, surveillance for people with F3 fibrosis detected through liver biopsy may also represent a cohort whose surveillance is cost-effective despite not yet having cirrhosis. Hepatic ultrasonography is used to monitor HCC every 4 to 8 months. The HCC’s quick 300-day median period for tumour doubling makes short interval surveillance necessary. [9] Between 65 and 80 percent of ultrasound surveillance is sensitive. [10] Ultrasound screening for HCC is ineffective in patients with end-stage liver disease, who may instead need cross-sectional imaging. This is based on a research of 27 people who underwent ultrasound examinations on average 90 days prior to liver transplantation, and it was discovered that ultrasound was only 20% sensitive to HCC after correlation with pathologic assessment of the liver explant. It is unclear if combining serum alphafetoprotein (AFP) measurement with ultrasound monitoring would be
advantageous. For illness identification, AFP is neither sensitive nor specific. This is due to the fact that many early HCCs do not release AFP and that AFP levels rise naturally as liver cirrhosis progresses. Before cirrhosis is identified, there is no reason to screen people with NAFLD for the disease. Contrary to virally induced cirrhosis, people with NAFLD-cirrhosis have a significantly lower incidence of HCC and overall fewer liver-related problems.

CURATIVE TREATMENT FOR HCC:
Partial surgical resection
Multinodular intrahepatic metastases are first caused by the metastatic spread of HCC through the portal vein, and extrahepatic metastases are then caused by the metastatic spread of HCC through the hepatic vein. This is why anatomic segmental resection along the portal blood supply is carried out in an effort to remove the HCC’s primary tumour while also catching hidden metastases in the portal vein. Multiple related contraindications exist when removing the liver (Table 1). The volume of the future in many circumstances.

Hepatic resection will be restricted by liver remnant (FLR) after resection. The FLR for cirrhotic individuals must equal at least 40% of the liver volume prior to resection. Many patients with an expected small FLR will have their portal vein embolized prior to resection, which results in ischemia of the embolized lobe and concurrent regenerative hypertrophy of the contralateral lobe (the FLR). After portal surgery, partial resection may not always be possible. Vein embolization due to iatrogenic tumor progression in the non-treated lobe because of rapid shunting of blood flow. Although this is less common with HCC than with metastatic disease, it remains a potential adverse outcome.

For this reason, Y90 may offer better outcomes for patients with small FLR as it can be used to both treat ipsilateral HCC and additionally will cause hypertrophy of the FLR. After a hepatic resection, the survival rate is high, ranging from 87% to 97% after one year to between 35% and 74% after five years. [30,33-35] As 5-year survival for tumours >10 cm is between 27% and 40%, which is better than alternative therapy, tumour size alone is not a limiting criterion for hepatic resection. [36] Patients with bilobar HCC or portal hypertension, which are often exclusion criteria, may benefit from partial resection.

CONCLUSIONS
Treatment of HCC with curative aim, such as liver transplantation, partial liver resection, or percutaneous RFA, offers a significant improvement in patient survival when feasible. Locoregional therapy, such as TACE and Y90, is frequently safe and can be used in conjunction with surgical therapies. It is unknown if there is a benefit to Y90 prior to liver resection in terms of ipsilateral tumour control or contralateral liver remnant hypertrophy, logical substitute for many patients. The SARAH and SIRveNIB trials showed that Y90 was inferior to sorafenib, however the sorafenib arm had nearly twice as many side effects. The role of either TACE or Y90 in combination with sorafenib for advanced illness will need to be clearly defined in the future. It has been greatly anticipated to compare HCC treated just with sorafenib with HCC treated in combination with sorafenib and Y90 in the ongoing SORAMIC and STOP-HCC trials.

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