

# Comprehensive Interventional Radiology Guide for the Treatment of Hepatocellular Carcinoma (HCC) in the Liver

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**Abstract**—Hepatocellular carcinoma (HCC) is the most frequent primary malignant tumor of the liver and one of the leading causes of cancer-related mortality globally. Because of its silent onset and frequent association with chronic liver disease, early detection is still challenging. Closely connected with viral hepatitis, metabolic problems, and cirrhosis, the global prevalence of HCC continues to climb, making it a major public health concern. Because it offers minimally invasive, image-guided, targeted, and organ-preserving therapies that minimize systemic toxicity and provide accurate tumor-directed treatment, interventional radiology (IR) has emerged as a crucial part of multidisciplinary HCC management. These therapies are particularly beneficial for patients who are poor surgical candidates and for those requiring downstaging or bridging prior to liver transplantation<sup>[1-2]</sup>

This comprehensive guide reviews essential liver anatomy and physiology, explores the etiopathogenesis and clinical manifestations of HCC, and provides an updated overview of diagnostic strategies and interventional therapies, including TACE, DEB-TACE, TARE, RFA, MWA, cryoablation, IRE, PVE, and combination approaches. Special attention is given on patient selection, imaging evaluation, operative approaches, complication management, and post-treatment assessment. This guidebook provides clinicians and trainees with an organized, evidence-based approach targeted at enhancing outcomes and standardizing interventional therapy for patients with liver cancer by including the most recent research, current guidelines, and useful insights<sup>[3]</sup>

**Index Terms**— Hepatocellular carcinoma (HCC), Interventional radiology. Transarterial chemoembolization (TACE), Drug-eluting bead TACE (DEB-TACE), Yttrium-90, Radiofrequency ablation (RFA), Microwave ablation (MWA), Cryoablation, Portal vein embolization (PVE), Downstaging and bridging, Liver transplantation, Multimodality therapy, Treatment response assessment.

Hepatocellular carcinoma (HCC) accounts for the majority of primary liver malignancies and remains a major worldwide health challenge due to its increased incidence, late-stage presentation, and limited treatment options. Chronic liver injury is intimately connected with alcoholic liver disease, non-alcoholic steatohepatitis (NASH), metabolic dysfunction-associated fatty liver disease (MASLD), non-alcoholic fatty liver disease (NAFLD), and viral hepatitis (HBV and HCV). Even with improvements in surveillance, many patients arrive with intermediate or advanced illness, making surgical resection or transplantation unsuitable. By offering accurate, reproducible, organ-preserving treatment options that increase survival and enhance quality of life, developments in imaging and interventional radiology have revolutionized the management of HCC<sup>[1, 4]</sup>

Interventional radiology (IR), which provides minimally invasive, tumor-directed treatments for both curative and palliative purposes, has emerged as a key component of multidisciplinary HCC therapy. Transarterial chemoembolization (TACE), drug-eluting bead TACE (DEB-TACE), transarterial radioembolization (TARE/Y-90), and percutaneous ablative modalities like radiofrequency and microwave ablation, cryoablation, irreversible electroporation, and portal vein embolization (PVE) are important IR techniques. IR treatments are critical not only for primary tumour control but also for down staging advanced disease, bridging suitable patients to liver transplantation, and combining with systemic medications in a multimodal therapeutic framework. These methods reduce systemic toxicity, permit follow-up therapies

## I. INTRODUCTION

when needed, and offer alternatives to patients who are not good candidates for surgery.<sup>[5-6]</sup>

Clinicians and trainees need a concise, evidence-based resource that summarizes current recommendations, procedural principles, patient selection criteria, and post-treatment assessment procedures as the therapeutic landscape continues to change. This comprehensive guide aims to provide an organized and accessible overview of IR approaches for HCC, covering current breakthroughs, technical issues, complication management, and future directions in image-guided oncologic therapy. The publication uses this paradigm to facilitate effective clinical decision-making in the treatment of liver cancers and to encourage standardized, high-quality interventional therapy.

## II. AIMS AND OBJECTIVES

The major objective of this guide is to provide a complete overview of liver anatomy and physiology covering segmental structure, vascular supply, and critical processes such as metabolism and detoxification essential for interpreting HCC development and executing image-guided therapies. It also seeks to explain the pathophysiology, clinical manifestation, and genesis of HCC, emphasizing risk factors such as cirrhosis, alcoholism, hepatitis, and metabolic disorders and how they influence the development and spread of tumors. Reviewing diagnostic techniques such as ultrasound, CT, MRI, CEUS, biomarkers, and biopsies, as well as staging systems like BCLC, TNM, Child-Pugh, and LI-RADS that direct treatment selection, is another goal<sup>[7-9]</sup>

All of the major interventional radiology treatments TACE, DEB-TACE, TARE, RFA, MWA, cryoablation, IRE and PVE as well as their indications, methods, results, and limits are covered in the handbook. Ultimately, it provides as a practical, therapeutically valuable resource for students, clinicians, and interventional radiologists to assist effective, evidence-based HCC therapy<sup>[10-11]</sup>

## III. METHODS AND MATERIALS

This narrative review was generated by a methodical assessment of existing research and clinical guidelines relating to the diagnostic and

interventional radiological therapy of hepatocellular carcinoma (HCC). A targeted literature search was undertaken utilizing major medical databases, and reference lists from key articles were personally examined to ensure inclusion of relevant studies. The indications, methods, and clinical efficacy of interventional radiology procedures, such as TACE, DEB-TACE, TARE, RFA, MWA, cryoablation, IRE, and PVE, were assessed<sup>[12-13]</sup>

The American Association for the Study of Liver Diseases (AASLD), European Association for the Study of the Liver (EASL), National Comprehensive Cancer Network (NCCN), Society of Interventional Radiology (SIR), and the Liver Imaging Reporting and Data System (LI-RADS) were among the major professional societies whose guidelines were incorporated. These resources were synthesized to give concise, evidence-based ideas relevant to contemporary HCC management<sup>[14-15]</sup>

**Morphology of Hepatocellular Carcinoma (HCC)**  
Hepatocellular carcinoma (HCC) exhibits a wide morphological spectrum that reflects its biological behaviour, degree of differentiation, and the underlying hepatic disease. HCC manifests grossly in three traditional ways: diffuse/infiltrative, nodular, and massive.

- The nodular type, most usually found in cirrhotic livers, appears as well-circumscribed lesions that may be encapsulated or non-encapsulated.
- The enormous type comprises of a huge main tumor that may coexist with smaller satellite nodules.
- The diffuse or infiltrative pattern spreads extensively over the liver parenchyma, often infiltrating the portal venous system, and is often linked with a poorer prognosis<sup>[16-17]</sup>

Under a microscope, HCC arises from malignant hepatocytes and displays a variety of architectural configurations, including as scirrhou, compact, trabecular, and pseudo glandular (acinar). The trabecular variation is most prevalent and is characterized by thicker hepatocyte plates separated by sinusoid-like vascular channels. Significant cytological atypia, nuclear pleomorphism, large nucleoli, elevated mitotic activity, and a disturbed reticulin framework are characteristics of poorly differentiated malignancies. Despite being malignant, HCC frequently maintains hepatocytic characteristics such as bile production, eosinophilic

cytoplasm, Mallory-Denk bodies, or fatty alteration. Vascular invasion, particularly of the portal or hepatic veins, is a distinguishing physical feature of aggressive illness and closely corresponds with unfavorable outcomes<sup>[18-19]</sup>

From the standpoint of imaging pathology correlation, HCC is a noticeably hypervascular tumor that receives most of its blood supply from the hepatic artery. This arterial predominance produces the characteristic radiological signal of arterial-phase hyperenhancement followed by washout in the portal venous or delayed phases—a critical need for non-invasive diagnosis. Tumor heterogeneity, including areas of necrosis, hemorrhage, fatty degeneration, and fibrosis, further alters imaging appearance and predicts different responses to locoregional therapy<sup>[20]</sup>

A thorough understanding of the morphological spectrum of HCC is crucial for accurate diagnosis, staging, and appropriate selection of interventional radiology treatments such as transarterial chemoembolization (TACE), transarterial radioembolization (TARE), irreversible electroporation (IRE), and cryoablation.

#### Causes of Hepatocellular Carcinoma (HCC)

Hepatocellular carcinoma (HCC) develops as the final result of persistent liver damage, inflammation, and fibrosis leading to cirrhosis. Major causes include chronic viral hepatitis, where HBV induces both direct carcinogenic effects and chronic inflammation, while HCV promotes carcinogenesis through steatosis, oxidative stress, and fibrosis. Alcohol-induced liver disease contributes through hepatocyte injury and alcoholic cirrhosis. NAFLD/MASLD, fuelled by obesity and diabetes, is an increasingly common etiology that may develop to HCC even without cirrhosis. Cirrhosis, irrespective of origin, remains the biggest risk factor<sup>[21-22]</sup>

Environmental causes include aflatoxin B1, which generates TP53 mutations and synergizes with HBV. Genetic and metabolic disorders such as hemochromatosis, Wilson's disease, and alpha-1 antitrypsin deficiency raise long-term risk. Chronic inflammation increases the likelihood of autoimmune and cholestatic illnesses (AIH, PBC, PSC). Smoking, obesity, diabetes, and ongoing

exposure to industrial pollutants or arsenic are further factors<sup>[23]</sup>

#### Symptoms of Hepatocellular Carcinoma (HCC)

Hepatocellular carcinoma (HCC) is generally asymptomatic in early stages, particularly in people with chronic liver disease. When the tumor grows larger, invades vascular structures, or exacerbates underlying cirrhosis, symptoms typically appear. Early systemic signs include accidental weight loss, anorexia, weariness, malaise, and low-grade fever. Patients may develop palpable masses, fullness, early satiety, right upper-quadrant abdominal pain, and ascites-related abdominal distension as the tumor progresses<sup>[24-25]</sup>

Symptoms related to liver failure and cirrhosis include jaundice, progressive ascites, peripheral edema, hepatic encephalopathy, and gastrointestinal bleeding from portal hypertension. Acute abdominal discomfort from a ruptured tumor or consequences like portal or hepatic vein thrombosis might be signs of advanced or aggressive illness. Paraneoplastic syndromes including hypoglycaemia, hypercalcemia, erythrocytosis, and thyrotoxicosis may also develop<sup>[26-27]</sup>

#### Diagnosis of Hepatocellular Carcinoma (HCC)

Hepatocellular carcinoma is diagnosed using a combination of laboratory markers, clinical evaluation, and distinctive imaging characteristics on multiphase contrast-enhanced modalities. Hepatocellular carcinoma (HCC) can often be discovered non-invasively based on imaging alone, without biopsy, in at-risk patients, particularly those with cirrhosis or chronic hepatitis B, according to AASLD and LI-RADS criteria.

##### 1. Clinical Evaluation:

Clinical examination is the primary method used to diagnose hepatocellular carcinoma (HCC), particularly in patients with existing liver disease. The first step is to identify risk factors because most cases of HCC occur in individuals with chronic hepatic impairment. Important risk factors include cirrhosis of any type, chronic hepatitis B or C infection, excessive alcohol use, and metabolic liver diseases as NAFLD/MASLD. Determining these variables aids in assessing the likelihood that a detected liver lesion is indicative of HCC as well as the patient's surveillance requirements<sup>[28, 29]</sup>

Patients may appear with nonspecific complaints as inadvertent weight loss, right upper-quadrant

abdominal pain, jaundice, or gradual abdominal distension from developing ascites, therefore a comprehensive review of symptoms is crucial.



Fig1: Enhancement pattern of HCC at contrast-enhanced US

These symptoms often signal tumor development or decompensation of underlying cirrhosis<sup>[30,31]</sup>. Clinicians check for hepatomegaly, ascites, and outward manifestations of chronic liver disease (spider angiomas, palmar erythema, muscle wasting) during the physical examination. Findings reinforce clinical suspicion and warrant additional assessment with laboratory testing and imaging<sup>[32]</sup>.

## 2. Laboratory Diagnosis:

By evaluating liver function, determining viral etiologies, and providing biochemical indicators that supplement imaging, laboratory examination aids in the diagnosis of hepatocellular carcinoma (HCC). The most used biomarker, alpha-fetoprotein (AFP), is raised in 50–60% of HCC patients, which makes it helpful for treatment monitoring and surveillance but not for diagnosis. Greater specificity is provided by other markers, such as AFP-L3% and PIVKA-II (DCP), which indicate aggressive tumor behavior or aberrant protein production. These indicators work better together to identify HCC in its early stages<sup>[33]</sup>.

Tests for liver function, including as bilirubin, INR, albumin, and AST/ALT, are used to assess baseline hepatic state, identify decompensation, and direct staging and interventional radiology decisions<sup>[29, 34]</sup>. Assessment of underlying liver disease is important since most patients with HCC have cirrhosis or chronic hepatitis. Testing for HBV DNA, HBeAg, anti-HCV, and HCV RNA reveals active viral replication, establishes the cause, and provides information for antiviral treatment and surveillance<sup>[35]</sup>.

## 3. Imaging Modalities:

Hepatocellular carcinoma (HCC) is mostly diagnosed by imaging, particularly in patients with chronic liver disease. Without the necessity for a biopsy, HCC may frequently be identified non-invasively using distinctive features on contrast-enhanced imaging. Ultrasonography, contrast-enhanced ultrasonography, multiphase CT, and MRI with liver-specific contrast are the main techniques.<sup>[36]</sup>

### i) Ultrasound (US) and Contrast-Enhanced Ultrasound (CEUS)

The first-line method for monitoring HCC is ultrasound, which is advised for at-risk individuals every six months. It is inexpensive, readily accessible, and helpful in identifying newly formed liver nodules. Any lesion larger than 1 cm discovered on monitoring ultrasonography should be treated with an MRI or contrast-enhanced CT scan. Additionally, Doppler ultrasound evaluates vascular involvement, especially portal vein thrombosis, which could be a sign of tumor invasion.

By employing microbubble contrast agents to provide a real-time evaluation of tumor vascularity, contrast-enhanced ultrasonography (CEUS) improves diagnostic capacity. Hepatocellular carcinoma typically shows arterial phase hyperenhancement (APHE) followed by late (>60 seconds), modest washout to distinguish it from other localized liver lesions. CEUS is particularly helpful for clarifying unclear CT or MRI data because it offers dynamic, radiation-free evaluation without the risks of iodinated or nephrotoxic contrast agents.<sup>[37]</sup>

### ii) Multiphase Contrast-Enhanced CT

The ability to evaluate dynamic enhancement patterns throughout arterial, portal venous, and delayed phases with excellent spatial resolution makes multiphase contrast-enhanced CT an essential imaging modality for the diagnosis of hepatocellular carcinoma (HCC). The behavior of contrast within the lesion is an important diagnostic sign as HCC develops a mainly arterial blood supply. Radiologists can distinguish HCC from benign or ambiguous nodules that are frequently found in cirrhotic livers using this multiphasic technique.<sup>[37, 38]</sup>



Fig-2: Enhancement of HCC at contrast-enhanced CT scan

The hallmark of HCC on CT is arterial phase hyperenhancement (APHE), where the lesion shows higher enhancement than the surrounding liver parenchyma during the arterial phase. This illustrates the arterial neovascularisation of the tumor. In the subsequent portal venous or delayed phases, many HCCs exhibit washout, appearing relatively hypoattenuating because the contrast clears from the tumor faster than from normal liver tissue. Furthermore, certain tumors have an augmenting capsule, which is a smooth peripheral ring that corresponds to fibrous tissue in delayed phases. <sup>[36]</sup>

The lesion satisfies the LI-RADS LR-5 criteria when APHE, washout, and capsule appearance occur together, indicating a definitive HCC diagnosis with extremely high specificity. When these imaging features are available, clinicians particularly interventional radiologists can confidently move on with treatment planning without needing a biopsy, simplifying care and lowering procedure risks <sup>[39-40]</sup>

### iii) MRI With Liver-Specific Contrast

The most sensitive imaging technique for identifying early and small hepatocellular carcinoma (HCC), especially in patients with cirrhosis, is magnetic resonance imaging (MRI) using liver-specific contrast agents. By assessing both vascular behavior and hepatobiliary function, hepatocyte-specific drugs such as gadoxetate disodium (Eovist/Gd-EOB-DTPA) improve the capacity to differentiate benign nodules from malignant lesions. MRI's better soft-tissue contrast and multiparametric capabilities are crucial for early detection since early HCCs may lack the fibrous capsule or obvious vascular abnormalities seen on CT <sup>[41-42]</sup>

The arterial phase is where multiphasic MRI evaluation starts, and HCCs usually show arterial phase hyperenhancement, which reflects their enhanced vascular supply. In later portal venous or delayed phases, many HCC lesions exhibit washout and appear relatively hypointense in comparison to the surrounding liver tissue. There may also be a capsule-like appearance, which indicates fibrous tissue encircling the tumor. Since HCC frequently exhibits restricted diffusion as a result of high cellular density, diffusion-weighted imaging (DWI) adds additional diagnostic value <sup>[43]</sup>

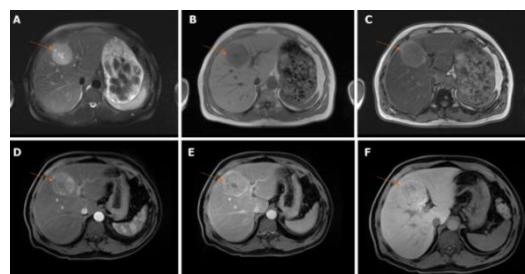


Fig3:Enhancement of HCC at contrast-enhanced MRI

The hepatobiliary phase, which is recorded about 20 minutes after contrast injection, is a crucial aspect of MRI with hepatobiliary contrast. HCC cells seem hypointense in comparison to normal liver parenchyma because they have fewer or no functional hepatocytes, which prevents them from absorbing Gadoxetate. This discovery is especially useful for locating tiny or unusual lesions that might not exhibit standard enhancing patterns <sup>[40]</sup>

When an observation shows restricted diffusion or hepatobiliary phase hypo intensity in addition to arterial hyperenhancement, washout, and/or capsule appearance, it meets the diagnostic criteria for definite HCC, LI-RADS LR-5. Clinicians can accurately diagnose HCC without a biopsy, regards to this classification's more than 95% specificity, allowing them to move straight to the proper interventional or surgical therapy. <sup>[44]</sup>

Treatment by Interventional Radiology (IR) Procedures:

A) Vascular intervention treatment

- i) Transarterial Chemoembolization (TACE)
- ii) Transarterial Radioembolization (TARE)
- iii) Portal Vein Embolization (PVE)

B) Non-vascular intervention treatment

- i) Radiofrequency Ablation (RFA)

- ii) Microwave Ablation (MW)
- iii) Irreversible Electroporation (IRE)
- iv) Cryoablation

#### A) Vascular Interventional Treatment

i) Transarterial Chemoembolization (TACE) :One of the most popular interventional radiology techniques for treating hepatocellular carcinoma (HCC) is transarterial chemoembolization (TACE), especially for patients who are not candidates for surgical resection or ablation. In addition to being utilized for downstaging or bridging patients awaiting liver transplantation, it is the standard of therapy for intermediate-stage HCC (BCLC Stage B) [45]

TACE takes advantage of the fact that HCC receives most of its blood supply from the hepatic artery, whereas normal liver tissue is primarily perfused by the portal vein. A catheter is carefully placed into the hepatic artery branches that supply the tumor, and then a mix of chemotherapy drugs—typically doxorubicin, cisplatin, or epirubicin—are delivered directly into the tumor. Embolizing particles that block arterial inflow, such as lipiodol, gelatin sponge, or drug-eluting beads, are then infused. [46]

Targeted chemotherapy and ischemia work together to cause tumor necrosis while protecting the adjacent liver parenchyma. Compared to conventional TACE (cTACE), drug-eluting bead TACE (DEB-TACE) delivers less systemic toxicity and better controlled, prolonged release of chemotherapy. [47].

TACE increases survival, lowers tumor burden, and is frequently repeated in cycles based on liver function and tumor response. For multifocal, incurable HCC without significant vascular invasion, it works particularly well. However, because patients with inadequate liver reserve, portal vein thrombosis, or decompensated cirrhosis are more likely to experience problems, cautious patient selection is necessary.

Overall, TACE is a fundamental minimally invasive treatment that is essential to the multidisciplinary care of HCC. It provides increased survival, local tumor control, and the possibility of downstaging toward curative therapeutic choices. [48]

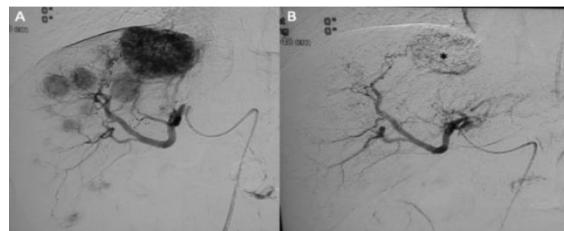


Fig4: TACE with doxorubicin and lipiodol for liver metastases; Image (A) of the right hepatic artery's digital subtraction angiography (DSA) reveals several hypervascular lesions in the liver; Image (B) DSA following TACE reveals lipiodol deposition in the lesions

#### ii) Transarterial Radioembolization (TARE):

Transarterial Radioembolization (TARE), also known as Selective Internal Radiation Therapy (SIRT), is a minimally invasive vascular interventional procedure used to treat hepatocellular carcinoma (HCC). It entails the direct introduction of microspheres containing radioactive isotopes, most frequently Yttrium-90 (Y-90), into the hepatic artery branches that feed tumors. TARE mainly uses internal radiation to kill cancer cells while protecting the surrounding liver tissue, in contrast to TACE, which causes ischemia [49-50]

TARE takes advantage of the fact that HCC is a hyper-vascular tumor, meaning that the hepatic artery provides the majority of its blood supply. Y-90 microspheres are infused as a microcatheter is carefully inserted into the arterial branch that supplies the tumor. With little exposure to systemic radiation, these microspheres cause localized tumor necrosis by lodging in the tumor microvasculature and releasing high-energy beta radiation over many days. [51].

Since radioembolization does not depend on arterial blockage and thus prevents worsening liver ischemia, one of the main benefits of TARE is its use in patients who are not candidates for TACE, such as those with portal vein thrombosis (PVT). Additionally, it works well for big, infiltrative, or multifocal HCC and can be used for bridging therapy while waiting for transplantation or for downstaging patients to transplant eligibility. [52].

Even in individuals with questionable liver function, TARE is well-tolerated since it typically results in fewer post-procedural symptoms than TACE. To evaluate lung shunting and avoid non-target embolization, however, meticulous pre-procedure

planning is crucial, including  $^{99m}\text{Tc}$ -MAA mapping. Overall, TARE provides targeted internal radiation therapy with superior tumor control, extended survival in certain patients, and a significant substitute in situations where chemoembolization or surgery is not practical. [53].

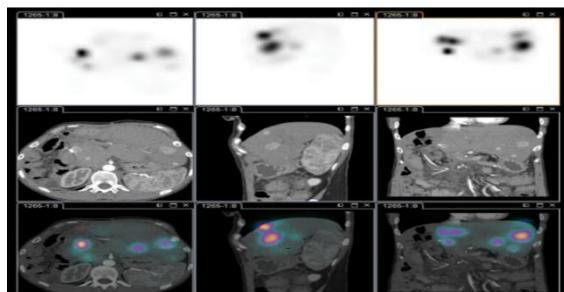


Fig 5:  $^{99m}\text{Tc}$ -MAA uptake in liver tumors. The SPECT images (top row), CT images (middle row), and fused SPECT/CT images (bottom row) demonstrate multiple liver tumors with radiotracer accumulation, aiding in pre-TARE mapping and assessment of tumor vascularity.

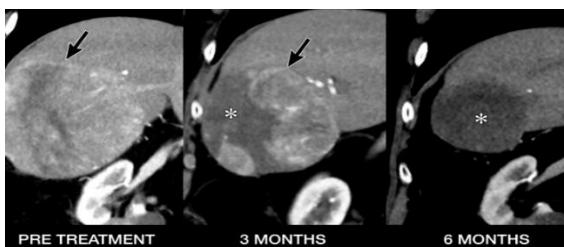


Fig 6: CT scan demonstrating tumor response after TARE. Baseline CT shows cirrhosis with hepatocellular carcinoma (arrow) involving liver segments V–VIII. The three-month post-treatment CT reveals substantial intratumoral necrosis, indicating a partial tumor response. Six months later, follow-up imaging demonstrates near-complete necrosis, consistent with a sustained and effective therapeutic response.

### iii) Portal Vein Embolization (PVE):

Portal vein embolization (PVE) is a vascular interventional radiology procedure that enhances the volume and function of the future liver remnant (FLR) in patients who require a major liver resection because of hepatocellular carcinoma (HCC). Many HCC patients have a low functional liver reserve, particularly those who have cirrhosis. Resection could be dangerous if there is not enough liver left after surgery. PVE assists in getting around this restriction. [54].

PVE functions by specifically obstructing the branches of the portal vein typically the right portal vein that supply the tumor-bearing areas of the liver. Over the course of two to six weeks, compensatory hypertrophy is stimulated by rerouting portal flow to the healthy liver segments by limiting blood flow to the damaged lobe. This increase in FLR lowers the risk of postoperative liver failure and makes hepatic resection safer. [55].

The PVE procedure is carried out percutaneously while being guided by fluoroscopic/CT and ultrasound. After inserting a needle or catheter into the portal venous system, embolic agents such as coils, polyvinyl alcohol (PVA) particles, n-butyl cyanoacrylate (NBCA) glue, or vascular plugs block the targeted portal branches. PVE is particularly useful when the tumor is limited to one lobe but the baseline liver reserve is insufficient for surgery. It is generally safe and has a low rate of complications. [56]

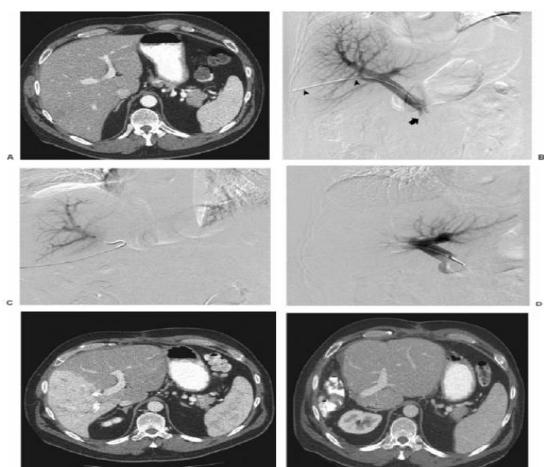


Fig 7: The entire process of portal vein embolization (PVE) using a transhepatic ipsilateral right-sided technique is shown in this sequence of images. A tumor-bearing right lobe with a borderline future liver residual (FLR) of around 25% is visible on pre-procedural contrast-enhanced CT (A). After percutaneous access, tris-acryl microspheres and coils are used for embolization after portal venography (B) and selective catheterization of the right portal branches (C). Segments V–VIII are confirmed to be occluded with left portal flow retained in the final portogram (D). A one-month follow-up CT scan (E) shows significant FLR hypertrophy to approximately 50%, and a post-hepatectomy CT scan (F) shows significant hypertrophy of the remnant liver, confirming good surgical preparation.

Considering all the factors, PVE is essential to the multidisciplinary therapy of HCC because it improves surgical results, increases the number of curative treatment options, and permits safe liver resection in patients who were previously unusable.

#### B) Non-vascular intervention treatment

i) Radiofrequency Ablation (RFA): A common minimally invasive thermal ablation method for treating early-stage hepatocellular carcinoma (HCC) is radiofrequency ablation (RFA). Surgical resection is considered a curative therapy option for patients who are not ideal candidates due to comorbidities, low liver reserve, or tumor location. Major guidelines (AASLD, EASL) support RFA as a first-line treatment for early-stage HCC; it works particularly well for small tumors ( $\leq 3$  cm). <sup>[57]</sup>

A needle electrode is percutaneously introduced into the tumor during RFA while being guided by CT or ultrasonography. The probe receives a high-frequency alternating electrical current, which causes frictional heat and coagulative necrosis of tumor tissue. The goal is complete ablation with a sufficient safety margin (about 5–10 mm) to reduce the likelihood of local tumor recurrence. <sup>[58]</sup>

Excellent local tumor control, minimal invasiveness, quick recovery times, and low complication rates are just a few benefits of RFA. It is particularly appropriate for individuals with compensated cirrhosis and single tumors in accessible areas. However, the effectiveness of RFA in tumors near major vessels may be limited by the heat-sink effect, in which blood flow absorbs thermal energy. Recurrence rates are higher in tumors larger than 3–5 cm; in these situations, RFA and TACE may be used to improve results. <sup>[59]</sup>

Overall, RFA is essential to the interventional management of HCC because it provides a safe, efficient, and organ-preserving option for therapy that, in carefully chosen patients, can reach survival rates comparable to surgical resection.

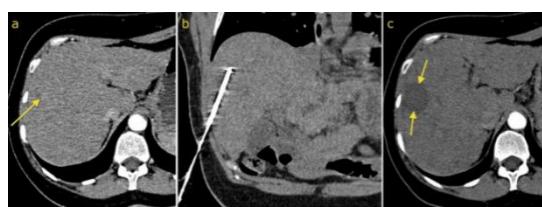


Fig 8: Image guided Radiofrequency Ablation (RFA) for Hepatocellular (HCC)

#### ii) Microwave Ablation (MW):

Microwave Ablation (MWA) is a minimally invasive, image-guided thermal ablation method that eliminates tumor tissue by using electromagnetic microwave energy to create intense heat. Hepatocellular carcinoma is commonly treated with it, particularly in patients who are not candidates for surgical resection or liver transplantation. Through a percutaneously inserted antenna placed into the tumor, MWA delivers microwave energy in the frequency range of 900–2450 MHz. This energy causes water molecules inside the tissue to oscillate quickly, resulting in high temperatures between 60°C and 150°C. This induces coagulative necrosis, irreversible tumor cell death, and destruction of the tumor microvasculature. MWA is more effective in burned, dehydrated, or aerated tissues and provides more consistent and uniform heating than radiofrequency ablation since it does not rely on tissue electrical conductivity. <sup>[60]</sup>

Under ultrasound, CT, or MRI guidance, the surgery is carried out under local anaesthesia with conscious sedation or, in certain situations, general anaesthesia. After precise tumor localization utilizing cross-sectional imaging, a microwave antenna is percutaneously inserted into the lesion's core. Microwave radiation is used for three to ten minutes, depending on the tumor's size and location. The objective is an ablation zone with a safety margin of 5–10 mm beyond the tumor margin. Imaging is done right away after ablation to verify total tumor removal and sufficient coverage. <sup>[61]</sup> MWA is most suited for patients with a single HCC or up to three tumors each  $\leq 3$ –4 cm, especially in early-stage illness (BCLC stages 0 and A). <sup>[62]</sup> Additionally, it is recommended for patients with compensated cirrhosis, poor surgical fitness, and recurring malignancies after surgical resection or transarterial chemoembolization (TACE). The advantages over RFA for tumors larger than 3 cm include faster heating, shorter operation periods, larger and more uniform ablation zones, reduced heat-sink effect, and fewer local recurrences. <sup>[63]</sup> The possibility of bile duct damage and technical challenges with subcapsular or hepatic dome lesions are among the limitations. The majority of issues are small and resolve on their own. About 90–98% of tiny lesions achieve complete tumor necrosis, and survival rates in early-stage HCC are similar to those of surgical excision. Additionally, MWA is

essential as a curative, combination, recurring, and bridging treatment for liver transplantation. [64]

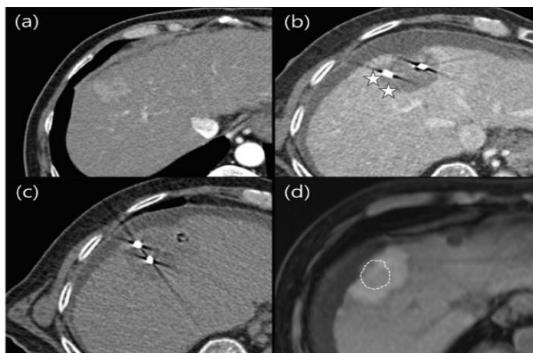


Fig 9: Patient with hepatocellular carcinoma (HCC) treated with microwave ablation (MWA). The pretreatment axial CT scan (a) shows a hypervascular HCC involving liver segment VIII. Following the initial ablation session, intraprocedural CT (b) demonstrates incomplete coverage of the tumor margin (arrows), indicating an inadequate ablative margin along the right side of the lesion. To achieve complete tumor coverage, additional ablation was performed by repositioning the MWA probe (c). A follow-up MRI obtained several months later (d) reveals a non-enhancing ablation zone with near-complete necrosis, confirming an adequate ablative margin and successful local tumor control. [65, 68]

### iii) Irreversible electroporation (IRE):

Hepatocellular carcinoma (HCC) is treated with Irreversible Electroporation (IRE), a non-thermal, image-guided ablation approach, especially when tumors are near vital tissues where heat-based ablation techniques like RFA or MWA present serious concerns. In contrast to thermal modalities, IRE uses many needle electrodes to deliver high-voltage, brief electrical pulses that permanently puncture tumor cell membranes. By disrupting cellular homeostasis and triggering apoptosis, this approach enables targeted tumor destruction while preserving the structural integrity of neighboring connective tissues. [65-66]

IRE is especially helpful for lesions that are close to important blood arteries, bile ducts, or the hepatic hilum, where the heat-sink effect can reduce the effectiveness of thermal ablation or where too much heat could harm important anatomical structures. Because IRE does not rely on heat energy and leaves vascular and ductal structures largely intact, it is a good treatment for tumors next to the portal

vein, hepatic veins, or central bile ducts. In order to assure accurate electrode placement and procedural safety, the treatment is carried out percutaneously under ultrasonography or CT guidance, usually needing general anesthesia and muscle relaxation. [67]

In carefully chosen patients, especially those with small HCC lesions (<3 cm) in physically problematic locations, clinical investigations show that IRE provides effective local tumor control. IRE greatly expands the therapy options for individuals who are not suitable candidates for thermal ablation or surgical resection, even if long-term outcome data are still developing. In general, IRE is a significant contribution to non-vascular interventional radiology therapies, allowing for the safe and efficient ablation of tumors close to critical structures while reducing collateral tissue damage. [65, 68]

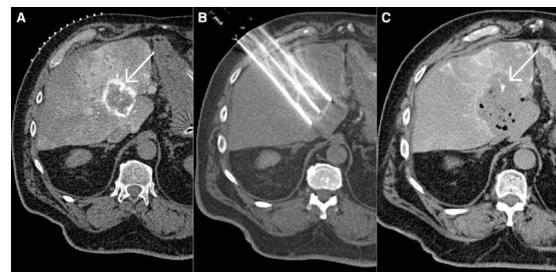


Fig 10: CT-guided irreversible electroporation (IRE) for HCC. (A) Arterial-phase CT shows a hyperenhancing hepatic tumor. (B) Multiple IRE electrodes are placed in parallel under CT guidance. (C) Post-IRE CT demonstrates a hypoattenuating ablation zone with intralesional gas while adjacent vessels remain patent, confirming successful non-thermal tumor ablation.

### iv) Cryoablation:

Cryoablation, a non-thermal, image-guided ablation technique, is increasingly used to treat patients with hepatocellular carcinoma (HCC) who are not candidates for surgery or thermal ablation. Unlike radiofrequency ablation (RFA) and microwave ablation (MWA), which utilize heat to destroy tissue, cryoablation uses extremely low temperatures to eliminate malignancies. This is accomplished by quickly freezing cryoprobes with pressurized argon gas (Joule-Thomson effect), creating a "ice ball" that surrounds the tumor and causes osmotic changes, cellular damage, ice crystal formation, microvascular damage, and eventually tumor necrosis. [69]

There are a number of benefits to cryoablation, particularly in delicate or complicated anatomical areas. Because the ice ball is directly visible under CT, MRI, or ultrasound, the danger of incomplete treatment is decreased and the ablation margin may be precisely controlled. Additionally, cryoablation is less painful than heat-based techniques and might be safer for lesions close to the liver capsule, diaphragm, biliary systems, or other nearby organs. Cryoablation can also be useful for tumors near major blood arteries since it is less impacted by the heat-sink effect.<sup>[70]</sup>

Clinical research demonstrates that cryoablation provides efficient local tumor control for small HCC lesions, with results in certain patients that are comparable to RFA. To increase efficacy in bigger or multifocal tumors, it can also be used in conjunction with transarterial therapies (such TACE). Although complications are rare, they can include bleeding, cryoshock, or damage to nearby organs, which highlights the importance of cautious patient selection and procedure monitoring. Generally, cryoablation is a useful non-vascular interventional radiology approach that increases the therapy choices for HCC by providing accurate, image-guided, minimally invasive tumor annihilation with good visibility and favorable safety in carefully chosen patients.<sup>[71-72]</sup>

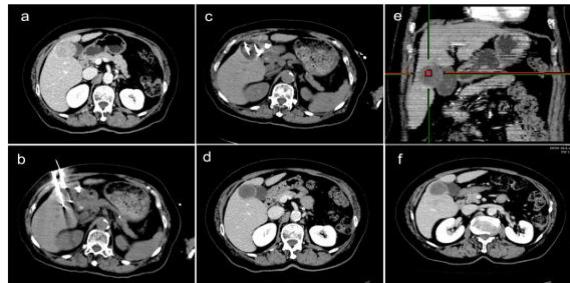


Fig 11: CT-guided percutaneous cryoablation of the liver demonstrating an HCC adjacent to the gallbladder. Baseline CT identifies the lesion (a), followed by CT-guided cryoprobe placement (b–c). Intraprocedural CT confirms adequate ice-ball coverage. Post-ablation imaging (d–e) shows a well-defined necrotic zone without gallbladder injury. Follow-up CT (f) demonstrates a non-enhancing ablation cavity, indicating successful tumor eradication.

#### IV. RESULTS

For hepatocellular carcinoma (HCC), interventional radiology-based therapies showed good clinical and

imaging results in both vascular and non-vascular therapy groups. TACE was one of the vascular therapies that effectively devascularized tumors, significantly reducing tumor burden and improving survival in patients with intermediate-stage illness, especially in multifocal, unresectable HCC without extensive vascular penetration. DEB-TACE demonstrated more prolonged intratumoral drug delivery and decreased systemic toxicity. Particularly the patient with portal vein thrombosis, TARE produced significant rates of intratumoral necrosis with long-lasting responses and was well tolerated with less post-embolization symptoms. Portal vein embolization (PVE) successfully increased future liver residual (FLR) volume (from ~25% to ~50%) to facilitate a safe major hepatectomy and reduce the risk of postoperative liver failure.<sup>[46, 27-73]</sup>

RFA and MWA both produced high complete ablation rates for early-stage HCC using non-vascular therapies; however, MWA showed bigger, more uniform ablation zones and a lower heat-sink effect, especially for tumors larger than 3 cm. For small lesions in anatomically difficult locations, IRE effectively controlled local tumors while protecting nearby arteries and bile ducts. IRE preserved nearby arteries and bile ducts while effectively controlling minor lesions in anatomically difficult areas. Overall, when properly chosen, IR treatments offered patients with HCC more curative alternatives, better survival results, downstaging and bridging to transplantation, and good local management.<sup>[57, 72, 74]</sup>

#### V. DISCUSSION

Interventional radiology (IR) is a key component of the multidisciplinary management of hepatocellular carcinoma (HCC), providing minimally invasive, liver-preserving treatment options across a broad spectrum of disease stages. Both vascular and non-vascular IR techniques are complementary, enabling effective local tumor control, improved survival, facilitation of curative surgery or liver transplantation, and expansion of treatment eligibility in individuals with limited hepatic reserve<sup>[27]</sup>

For intermediate-stage HCC (BCLC stage B), transarterial chemoembolization (TACE) is still the recommended vascular strategy. TACE allows for

the targeted delivery of chemotherapeutic drugs in conjunction with arterial embolization, which causes tumor ischemia and necrosis while maintaining non-tumorous liver parenchyma, by taking advantage of the preferential arterial blood supply of HCC. Drug-eluting bead TACE lessens systemic toxicity and enhances intratumoral drug retention. However, since patients with substantial vascular invasion or decompensated cirrhosis have worse outcomes, cautious patient selection is essential. An important alternative is transarterial radioembolization (TARE), especially for individuals with a large tumor burden or portal vein thrombosis. Yttrium-90 microspheres are delivered via TARE, which uses internal radiation instead of ischemia to control tumors with favorable tolerability and long-lasting effects. By causing the future liver residue to enlarge, portal vein embolization (PVE) has a unique vascular role that permits safe hepatic resection in individuals with inadequate baseline liver reserve. [55, 83-84, 86]

When treating early-stage HCC, non-vascular IR treatments are essential. For tumors  $\leq 3$  cm, radiofrequency ablation (RFA) offers excellent local control; however, the heat-sink effect may restrict its effectiveness. By creating larger, more consistent ablation zones and improving efficacy for tumors larger than 3 cm, microwave ablation (MWA) gets around a number of these restrictions. For lesions next to large vessels or bile ducts, irreversible electroporation (IRE) provides a non-thermal alternative that effectively controls the area while protecting important structures. By enabling accurate margin vision and safe ablation close to delicate anatomical features, cryoablation considerably increases the range of therapy possibilities. [58, 61, 67, 70]

In general, interventional radiology procedures are complementary rather than competitive. The importance of interventional radiology in the therapy of hepatocellular carcinoma in the modern era is highlighted by the requirement for customized treatment selection based on tumor features and underlying liver function.

## VI. CONCLUSION

Nowadays, interventional radiology (IR) is a crucial component of the multidisciplinary treatment of hepatocellular carcinoma (HCC). IR successfully

treats tumor burden across various disease stages while addressing the difficulties of reduced hepatic reserve by offering a wide range of minimally invasive, liver-preserving vascular and non-vascular procedures. These image-guided treatments enable customized treatment plans that strike a balance between liver function preservation and oncologic management.

In intermediate and advanced HCC, vascular treatments such as transarterial chemoembolization (TACE) and transarterial radioembolization (TARE) provide efficient locoregional tumor management, facilitating downstaging, bridging to transplantation, and enhancing survival for non-surgical patients. By causing the future liver residual to become larger, portal vein embolization (PVE) increases the number of treatment possibilities. Non-vascular techniques include radiofrequency ablation (RFA), microwave ablation (MWA), irreversible electroporation (IRE), and cryoablation provide curative or almost curative treatment for early-stage and specific complex lesions.

All things considered, IR modalities are complimentary rather than competitive, and the best results are obtained by customized patient selection based on liver function, tumor features, and therapeutic objectives.

## REFERENCE

- [1] Singh SP, Madke T, Chand P. Global epidemiology of hepatocellular carcinoma. *J Clin Exp Hepatol.* 2025;15(2):102446.
- [2] Yang JD, Hainaut P, Gores GJ, Amadou A, Plymoth A, Roberts LR. A global view of hepatocellular carcinoma: trends, risk, prevention and management. *Nat Rev Gastroenterol Hepatol.* 2019;16(10):589-604.
- [3] Davis CR. Interventional radiological treatment of hepatocellular carcinoma. *Cancer Control.* 2010;17(2):87-99.
- [4] Wong SW, Ting YW, Chan WK. Epidemiology of non-alcoholic fatty liver disease-related hepatocellular carcinoma and its implications. *JGH Open.* 2018;2(5):235-241.
- [5] Sangro B, Iñarriaga M, Bilbao JI. Radioembolization for hepatocellular carcinoma. *J Hepatol.* 2012;56(2):464-473.
- [6] Singh R, Makary MS. Locoregional therapies for hepatocellular carcinoma with portal vein

tumor thrombus. *J Gastrointest Cancer.* 2025;56(1):162.

[7] Chartampilas E, Rafailidis V, Georgopoulou V, Kalarakis G, Hatzidakis A, Prassopoulos P. Current imaging diagnosis of hepatocellular carcinoma. *Cancers (Basel).* 2022;14(16):3997.

[8] Chartampilas E, Rafailidis V, Georgopoulou V, Kalarakis G, Hatzidakis A, Prassopoulos P. Current Imaging Diagnosis of Hepatocellular Carcinoma. *Cancers (Basel).* 2022 Aug 18;14(16):3997. doi: 10.3390/cancers14163997. PMID: 36010991; PMCID: PMC9406360.

[9] Berman ZT, Newton I. Diagnosis, staging, and patient selection for locoregional therapy to treat hepatocellular carcinoma. *Semin Intervent Radiol.* 2020;37(5):441-447.

[10] Vande Lune P, Abdel Aal AK, Klimkowski S, et al. Hepatocellular carcinoma: diagnosis, treatment algorithms, and imaging appearance after transarterial chemoembolization. *J Clin Transl Hepatol.* 2018;6(2):175-188.

[11] Agnello F, Taibbi A, Galia M, et al. Hepatocellular carcinoma treatment response: imaging findings and criteria. *World J Radiol.* 2025;17(10):108804.

[12] Izzo F, Granata V, Grassi R, et al. Radiofrequency ablation and microwave ablation in liver tumors: an update. *Oncologist.* 2019;24(10):e990-e1005.

[13] Hao K, Paik AJ, Han LH, Makary MS. Yttrium-90 radioembolization treatment strategies for hepatocellular carcinoma. *World J Radiol.* 2024;16(10):512-527.

[14] Zhu D, Yuan D, Wang Z, Chen S. Efficacy of DEB-TACE combined with radiofrequency ablation versus DEB-TACE alone in hepatocellular carcinoma. *Medicine (Baltimore).* 2019;98(26):e15682.

[15] Xu Z, Xie H, Zhou L, Chen X, Zheng S. Combination strategy of transarterial chemoembolization and radiofrequency or microwave ablation against hepatocellular carcinoma. *Anal Cell Pathol (Amst).* 2019;2019:8619096.

[16] Zeng JY, Piao XH, Zou ZY, et al. Cryoablation with drug-loaded bead embolization in unresectable hepatocellular carcinoma. *Oncotarget.* 2018;9(7):7557-7566.

[17] Vij M, Calderaro J. Pathologic and molecular features of hepatocellular carcinoma: an update. *World J Hepatol.* 2021;13(4):393-410.

[18] Torbenson MS. Hepatocellular carcinoma: morphological heterogeneity, growth patterns, and subtypes. *Hum Pathol.* 2021;112:86-101.

[19] Schlageter M, Terracciano LM, D'Angelo S, Sorrentino P. Histopathology of hepatocellular carcinoma. *World J Gastroenterol.* 2014;20(43):15955-15964.

[20] Kim H, Jang M, Park YN. Histopathological variants of hepatocellular carcinoma according to WHO classification. *J Liver Cancer.* 2020;20(1):17-24.

[21] Cannella R, Dioguardi Burgio M, Beaufrère A, et al. Imaging features of histological subtypes of hepatocellular carcinoma: implication for LIRADS. *JHEP Rep.* 2021;3(6):100380.

[22] Kew MC. Hepatocellular carcinoma: epidemiology and risk factors. *J Hepatocell Carcinoma.* 2014;1:115-125.

[23] McGlynn KA, London WT. The global epidemiology of hepatocellular carcinoma: present and future. *Clin Liver Dis.* 2011;15(2):223-243.

[24] Hamed MA, Ali SA. Non-viral factors contributing to hepatocellular carcinoma. *World J Hepatol.* 2013;5(6):311-322.

[25] Chu YJ, Yang HI, Wu HC, et al. Aflatoxin B1 exposure increases the risk of hepatocellular carcinoma. *Eur J Cancer.* 2018;94:37-46.

[26] Rapisarda V, Loreto C, Malaguarnera M, et al. Hepatocellular carcinoma and occupational exposure. *World J Hepatol.* 2016;8(13):573-590.

[27] Forner A, Reig M, Bruix J. Hepatocellular carcinoma. *Lancet.* 2018;391(10127):1301-1314.

[28] Ginès P, et al. Ascites in cirrhosis. *N Engl J Med.* 2004;350:1646-1654.

[29] Tsochatzis EA, Bosch J, Burroughs AK. Liver cirrhosis. *Lancet.* 2014;383(9930):1749-1761.

[30] El-Serag HB. Hepatocellular carcinoma. *N Engl J Med.* 2011;365(12):1118-1127.

[31] Sarin SK, et al. Acute-on-chronic liver failure: consensus recommendations. *Hepatol Int.* 2014;8(4):453-471.

[32] Trevisani F, D'Intino PE, Morselli-Labate AM, et al. Serum alpha-fetoprotein for diagnosis of hepatocellular carcinoma. *J Hepatol.* 2001;34(4):570-575.

[33] Best J, et al. Combination of AFP, AFP-L3 and PIVKA-II improves diagnostic performance in HCC. *BMC Cancer.* 2020;20:1135.

[34] Kamath PS, Wiesner RH, Malinchoc M, et al. A model to predict survival in end-stage liver disease. *Hepatology*. 2001;33(2):464-470.

[35] Lok AS, McMahon BJ. Chronic hepatitis B: update 2009. *Hepatology*. 2009;50(3):661-662.

[36] Marrero JA, Kulik LM, Sirlin CB, et al. Diagnosis, staging, and management of hepatocellular carcinoma. *Hepatology*. 2018;68(2):723–750. doi:10.1002/hep.29913.

[37] Clouston M, et al. WFUMB-EFSUMB guidelines on CEUS in the liver. *Ultraschall Med*. 2013;34(1):11-29.

[38] Murphy A, Dabirifar S, Bell D. CT triple-phase liver protocol. *Radiopaedia.org*. Accessed Dec 5, 2025.

[39] Mitchell DG, et al. LI-RADS: CT and MRI diagnostic criteria for HCC. *J Magn Reson Imaging*. 2014;40(3):566-580.

[40] Moura Cunha G, Chernyak V, Fowler KJ, Sirlin CB. Up-to-Date Role of CT/MRI LI-RADS in Hepatocellular Carcinoma. *J Hepatocell Carcinoma*. 2021 May 31;8:513-527. doi: 10.2147/JHC.S268288. PMID: 34104640; PMCID: PMC8180267.

[41] Liu X, Jiang H, Chen J, et al. Gadoxetic acid-enhanced MRI vs MDCT for small HCC: meta-analysis. *Liver Transpl*. 2017;23(12):1505-1518.

[42] Ichikawa T, Sano K, Morisaka H. Diagnosis of pathologically early HCC with EOB-MRI. *Liver Cancer*. 2014;3(2):97-107.

[43] Kim YK, et al. Gadoxetic acid-enhanced MRI for diagnosing HCC. *Radiology*. 2019;292(3):685–694.

[44] Koh DM, Collins DJ. Diffusion-weighted MRI in oncology. *AJR Am J Roentgenol*. 2007;188(6):1622-1635.

[45] Yao FY, Kerlan RK Jr, Hirose R, et al. Downstaging HCC prior to liver transplantation. *Hepatology*. 2008;48(3):819-827.

[46] Llovet JM, Real MI, Montaña X, et al. Chemoembolization vs symptomatic treatment in unresectable HCC. *Lancet*. 2002;359(9319):1734-1739.

[47] Lo CM, Ngan H, Tso WK, et al. Randomized trial of TACE for unresectable HCC. *Hepatology*. 2002;35(5):1164-1171.

[48] EASL-EORTC. Clinical practice guidelines: management of hepatocellular carcinoma. *J Hepatol*. 2012;56(4):908-943.

[49] Sacco R, Conte C, Tumino E, et al. Transarterial radioembolization for hepatocellular carcinoma. *J Hepatocell Carcinoma*. 2016;3:25-29.

[50] Salem R, Mazzaferro V, Sangro B. Yttrium-90 radioembolization for hepatocellular carcinoma. *Hepatology*. 2013;58(6):2188-2197.

[51] Lee HM, Alder L, Nguyen M, et al. Long-term outcomes of Y90 radioembolization in HCC. *J Gastrointest Oncol*. 2023;14(3):1378-1391.

[52] Yang Y, Si T. Y-90 radioembolization vs TACE: meta-analysis. *Cancer Biol Med*. 2018;15(3):299-310.

[53] Kallini JR, Gabr A, Salem R, Lewandowski RJ. Transarterial radioembolization with yttrium-90. *Adv Ther*. 2016;33(5):699–714. doi:10.1007/s12325-016-0324-7.

[54] Farges O, Belghiti J, Kianmanesh R, et al. Portal vein embolization before right hepatectomy. *Ann Surg*. 2003;237(2):208-217.

[55] Abulkhir A, Limongelli P, Healey AJ, et al. Preoperative portal vein embolization: meta-analysis. *Ann Surg*. 2008;247(1):49-57.

[56] Madoff DC, Abdalla EK, Vauthey JN. Portal vein embolization before hepatic resection. *J VascIntervRadiol*. 2005;16(6):779-790.

[57] Huang J, Yan L, Cheng Z, et al. Radiofrequency ablation vs resection for HCC. *Ann Surg*. 2010;252(6):903-912.

[58] Chen MS, et al. Ablation versus resection for small HCC. *Gastroenterology*. 2006;130(7):2013-2024.

[59] Bruix J, Sherman M. Management of hepatocellular carcinoma: an update. *Hepatology*. 2011;53(3):1020–1022. doi:10.1002/hep.24199.

[60] Poggi G, Tosoratti N, Montagna B, Picchi C. Microwave ablation of hepatocellular carcinoma. *World J Hepatol*. 2015;7(25):2578-2589.

[61] Sun AX, Cheng ZL, Wu PP, et al. Microwave ablation for medium-sized HCC. *World J Gastroenterol*. 2015;21(10):2997-3004.

[62] Jiang L, Liang C, Xie F, Zheng Y. Microwave ablation in solitary HCC. *Hepatobiliary SurgNutr*. 2023;12(4):622-624.

[63] Facciorusso A, Di Maso M, Muscatiello N. Microwave vs radiofrequency ablation for HCC. *Int J Hyperthermia*. 2016;32(3):339-344.

[64] Dou JP, Yu J, Yang XH, et al. Microwave ablation near large vessels. *Oncotarget*. 2017;8(17):28758-28768.

- [65] Kingham TP, Karkar AM, D'Angelica MI, et al. Irreversible electroporation for hepatic tumors. *J Am Coll Surg.* 2012;215(3):379-387.
- [66] Davalos RV, Mir IL, Rubinsky B. Tissue ablation with irreversible electroporation. *Ann Biomed Eng.* 2005;33(2):223-231.
- [67] Scheffer HJ, Nielsen K, de Jong MC, et al. Irreversible electroporation: systematic review. *J VascIntervRadiol.* 2014;25(7):997-1011.
- [68] Sutter O, et al. Safety and efficacy of irreversible electroporation in HCC. *Radiology.* 2017;284(3):878-886.
- [69] Hu KQ. Advances in cryoablation therapy for liver tumors. *J Clin Gastroenterol.* 2014;48(10):830-836.
- [70] Zhang W, Wang Y, Zhao X, et al. CT-guided cryoablation for high-risk HCC. *AcadRadiol.* 2024;31(11):4434-4444.
- [71] Wang C, et al. Cryoablation vs radiofrequency ablation for HCC. *Clin Gastroenterol Hepatol.* 2015;13(8):1423-1431.e1.
- [72] Kim GM, Won JY, Kim MD, et al. Cryoablation of high-risk hepatocellular carcinoma. *Cardiovasc InterventRadiol.* 2016;39(10):1447-1454.
- [73] Lammer J, Malagari K, Vogl T, et al. PRECISION V study: DEB-TACE for HCC. *Cardiovasc InterventRadiol.* 2010;33(1):41-52.
- [74] Salem R, Lewandowski RJ, Mulcahy MF, et al. Radioembolization for hepatocellular carcinoma. *Gastroenterology.* 2010;138(1):52-64.