Q. Role of various combination of tumor markers in diagnosis of HCC ?
A. As per the guidelines there is no role of tumor makers in diagnosis of HCC. In cirrhotic patients diagnosis is based on imaging characteristics and in non-cirrhotic liver its based on histopathology. Combined detection of tumor markers can increase diagnostic rate but they are not cost effective and have false positivity also.

Q. Role of external beam radiotherapy in treatment of HCC?
A. role of EBRT is found in subset of patients who have tumor thrombosis. Various studies has shown response rate ranging from 10 to 70%. More evidence needed before recommendation.

Q. Why HCC in HCV has better prognosis than HBV HCC though HCV HCC occurring in cirrhosis?
A. HBV is a oncogenic virus. Its get integrated with host DNA and forms various quasispecies and undergo lots of mutations. That's why HBV can cause HCC without cirrhosis however HCV can cause HCC only if there is high fibrosis(F3 and above).

Q. What is the role of peptide vaccine in preventing HCC?
A. Not a proven role. Only few studies has done which has targeted glypican-3 as it is found to be most specific for HCC which has shown some effect.

Q. Role of multiphase CT abd for survillance for HCC ?
A. MDCT is not cost effective for surveillance. MDCT also have radiation risk and use of contrast agents are also causes risk for nephropathy.

Q. What is the role of nanoparticle technology in HCC?
A. Nanoparticle technology uses nanoparticles to deliver antitumor drugs at the target lesion. They enhances the efficacy of chemotherapeutic drugs like doxorubicin. Further studies are needed.

Q. Sir, in Hepatitis B positive without cirrhosis, from when scrrescre of HCC to be initiated? Patient of HBsAg non cirrhotic, low HBV DNA, having HCC, antiviral are indicated?
A. Asian male >40, female> 50 and family history of HCC should be screened. HBV DNA level is a strong predictor of development of HCC, if HBV DNA is low then HCC risk is low, even if patient develop HCC, other factors should also be sought like HCV plus HBV, NASH. If they develop HCC treatment should be started to prevent reactivation.

Q. Role of TACE in HCC occuring in BCS without cirrhosis?
A. Secondary BCS can occur in HCC for which TACE is effective treatment. If primary BCS develop HCC that usually happens after the development of cirrhosis. Non cirrhotic BCS patients developing HCC is rare and even if it occurs surgery and TACE are effective options.

Q. Is there a need to do AFP for surveillance of HCC in a non-cirrhotic patient with portal vein thrombosis?
A. Not cost effective. DCP is a marker which predict development of tumor thrombosis but again combining tumor markers are found to be not cost effective and also have false positivity.

Q. Role of DCP in diagnosing hcc ??? And does DCP commercially available ???

A. Already described. It is available in selected centers and labs.

Q. dysplastic nodule on CECT with elevated PIVKA and normal AFP , how to proceed
A. if it is less than 1 cm nothing to be done and repeat imaging after 4 months. If it is larger than 1 cm. triple phase ct or mri with hepatospecific contrast should be done.

Q. Is there any role of aspirin in prevention of HCC in HBV and HCV patients?
A. Recent NEJM study has shown 30% risk reduction.

Q. How to differentiate a dysplastic nodule from HCC without tissue diagnosis?
A. Triple phase CT/MRI/CEUS showing characteristic features of HCC that is Non rim arterial hyperenhancement and portal venous washout. MRI with hepatospecific contrast can tell about the high risk dysplastic nodule which can be followed with imaging.

Q. Why does AFP rise in HCC Has AFP any significance in intrahepatic cholangiocarcinoma
A. AFP is normally produced during fetal development by the liver and yolk sac. In HCC malignant hepatocyte produce it in large quantity. It can also be produced by regenerating hepatocytes and inflammation

Q. CHB patient with mild elevations of transamnitis with elevated AFP 150ng/ml. USG showing no focal mass lesion in liver. TPCT abdomen no mass lesion. HBV DNA <2000IU/ml. How to approach these patients.whethet to attribute AFP to hbv or very small HCC
A. Inflammation can leads to raised AFP. if CT/MRI doesn't show any lesion that nothing to be done. 6 monthly USG

Q. In cirrhotics having Liver Sol,having deranged rft , which is the best modality.cect vs mri.any cut off of serum creatinine.
A.. MRI/CEUS

Q. What is the added advantage of AFP plus afpL3 and PIVKA for HCC detection. Can this increase the sensitivity in HCC screening
A. They increase the sensitivity but with the cost of false positivity and studies has shown that they are not cost effective for surveillance.

Q. My relative is HCV related cirrhosis child A and MELD 11 AFP 170, USG no SOL but CT dynamic view showing 9mm ?dysplatic nodule.. what to do next? Should we do MRI now or should we wait for one month then to recheck AFP?
A. USG at 4 month. If any growth change get done CT/MRI.

Q. Sir, can we use BCLC classification in non cirrhotic HCC as iBCLC requires CTP score
A. BCLC is for cirrhotic patients. In non cirrhotic patients evaluate for surgical candidacy.

Q. Is indirect markers of portal hypertension like portal vein diameter or varices on endoscopy an absolute contraindication for curative resection in hCC?
A. Not a absolute contraindication but post surgery liver decompensation risk will be high.

Q. While classifying asBCLC class C, do portal vein invasion and thrombosis imply the same?
A. Portal Vein tumor thrombosis

Q. What is the role of PIVKA in HCC survillance?
A. not recommended for surveillance. It corelated with tumor thrombosis or advanced liver cancer.

Q. Which is the better staging system to be used in India- BCLC and HKLC
A. BCLC is validated well and most commonly used system for HCC staging.

Q. How do we assess the mortality rate preoperative stage? What actually means mortality <3%
A. we assess risk of decompensation. But to say optimal surgical candidacy it was defined as that perioperative mortality should be < 3%

Q. When do we prefer MWA to RFA sir?
A. when the tumor location is near the blood vessel, subcapsular or near the bowel.

Q. for India , do u think we need other staging than BCLC
A. BCLC is a bit conservative while categorising treatment option, that's why the role of multidisciplinary team is important.

Q. What is the risk of tumour seeding in early liver lesion(<2cm) biopsy? would diagnostic plus therapeutic resection be a better option here?
A. 0.5%. diagnostic resection is not recommended because of risk of decompensation and very accurate diagnostic modalities in form of imaging.

Q. In case of large (8-10cm) exophytic HCC can TACE be done?
A. HCC > 10 cm is a relative contraindication for TACE. 8-10 cm exophytic lesion will have high risk of tumor rupture and dissemination.

Q. Role of voulmetry in resection. Cut off for resection? Thank you sir
A. volumetric assessment is helpful to predict post surgery outcomes. Usually 30 to 40 % of Functional liver reserve should be there after surgery to meet the demand of body.

Q. Dawnstaging protocol in management of HCC
A. Can use locoregional therapy like TACE, TARE, SBRT for downstaging. Target of 100% tumor necrosis however in only 30% of cases it could be achieved. Prognosis after TACE can be predicted by HAP score.

Q. Any role of IHC in diagnosis of HCC
A. IHC has a important role to distinguish combined HCC/CC from HCC due to the different therapeutic modalities, when the imaging features are not typical. Expression of CK 19 has shown poor prognosis.

Q. Should we use CU HCC criteria for surveillance of hcc in chronic hepatitis b?
A. need fmore studies for proper validation of CU HCC criteria

Q. when to start hcc surveillance in alcoholic and NASH cirrhosis?
A. after the development of cirrhosis criteria is to do USG 6 monthly.

Q. Diagnostic algorithm for fibrolamellar ? Is biopsy mandatory?

A. biopsy is gold standard but investigation of choice is imaging in form of MDCT which showed typical features of fibrolamellar HCC with central scar which is hypointense on both T1 and T2.

Q. Why we are not labelling <1cm lesion as HCC in spite of very short window for therapeutic approach ? Why should we wait to it becomes >2cm size?
A. Because < 1 cm diagnosis of HCC is very difficult even with biopsy and treatment outcomes are not different than 2 cm lesion

Q. Legal aspeiof starting treatment without biopsy???
A. Not any. Because all the guidelines suggestive of image based diagnosis in cirrhotic liver.

Q. What is maximum size cut off rfa can b taken
A. 3 cm. beyond that it should be combined with other modality like TACE.

Q. Is there any difference outcome with ablative modalities in deeper lesions ?
A. Deeper lesions can be treated with ablative therapy but for RFA tumor should not be near to the blood vessel or hilum(Heat sink effect)

Q. Are NASH and chronic hepatitis C non-cirrhotics to be screened for HCC ?
A. Hep C with advanced fibrosis (F3) should be screened. For NASH it is debatable.

Q. Do we need screening for patient with cirrhosis whose performance status is two or say 3 due to any reason
A if patients has lots of comorbidities and poor performance status > 2 and poor liver function , Screening should not be done .

Q. How to differentiate between dysplastic nodule, regenerative nodule and HCC on triphasic CT scan?
A. regenerative nodules are non enhancing in arterial phase and might have fibrous septa surrounding it. Dysplastic nodules are arterial hyperenhancing however doesn't washout on portal phase. HCC has typical early arterial hyperenhancement and portal venous washout.

Q. How to differentiate pseudo progression and hyperProgression in HCC treatment
A. No gold std yet, however use of liquid biopsies, tumor markers and liver biopsy can differentiate them.

Q. Sir preserve liver function is Child criteria or functional reserve based
A. CTP A, CTP B upto 8 without ascites

Q. In HCV related cirrhosis after SVR with antiviral, how often you should screen for HCC.
A. Once cirrhosis, USG at 6 monthly interval.

Q. Case of 25 yr old male with Chronic HBV hepatitis with multifocal large HCC with ECOG 0, CTP-5/6 what are the treatment options?
A. Rule out extrahepatic mets, vascular invasion, if not best candidate for LT.

Q. Sir, What should we do in a case of biopsy proven dysplastic nodule ?

A. most of the dysplastic nodules remain unchanged or may disappear. If high grade dysplasia is there and nodule is growing on follow up imaging. One can go for CT/MRI with hepatospecific contrast or can treat it with RFA/LR.

Q. For patient without cirrhosis upto which size surgery is curative In hcc?
A. Any size can be treated with surgery in non cirrhotics provided remaining adequate liver volume.

Q. HCC surveillance after DAA for hepatits c cirrhosis should be more frequently?
A. No. systemic revies has shown that DAA don't increase the risk of HCC.

Q. Is typical imaging features on imaging enough for diagnosis of hcc in non cirrhosis settings
A. imaging features are same as in cirrhotic liver however tissue diagnosis is required for labelling as HCC in non cirrhotic patients.

Q. What about okuda staging instead of bclc ???
A. Okuda doesn't address small tumors efficiently. Its separates tumors in two category whether they are less or mre than 50% of liver volume. BCLC is more advanced than okuda.

Q. A big problem in HCC is the large gap between treatment options and the number of patients subjected to treatment. Majority of patients at our centre come at intermediate or advanced stage. They dont agree for TACE/ TARE/ Transplant due to financial constraints and limited life expectancy.Do you feel that in the coming decade this gap will be reduced. What measures you feel can help in reducing this gap?
A. Effective surveillance system can detect cancer at an early stage which should be implemented effectively. In future various drugs including immunotherapies has shown a promising treatment outcomes but again the cost effectiveness of these drugs would be a problem.

Q. Which is the preferred sub classification of BCLC B-kinki or bolondi
A. Kinki classification is modified bolondi classification which is similar to the Bolondi's substaging system to a certain extent, but Kinki criteria is simpler and easier to apply, and resection and even ablation are included as treatment options while in bolondi only TACE has been recommended. Both needs to be validated in large cohort.

Q. what is your personal experience regarding transplant advised by HKLC in CTP C patients ??
A. On selective cases if performance status is good transplant can be considered. Liver transplant should be sought my multidisciplinary team wherever possible, specially in the cases where the reason for high CTP score is temporary like Hep A or E infection, low albumin because of acute phase reactant.

Q. Caveat of fibroscan role in diagnosis of cirrhosis in presence of hcc
A. can be falsely high LSM.

Q. Precautions in using immune checkpoint inhibitors in post transplant metastases? As these drugs stimulate T cells whereas patient needs immunosuppression to prevent rejection.

A. Role of ICI in post transplant HCC is not clear. Multiple studies has shown around 25% rejection of graft after ICI and more common for nivolumab than pembrolizumab. It occurs as an early adverse event mostly within 2 to 3 weeks.

Q. What is the role of combined modality treatment in HCC like sorafenib with TACE/RFA ? any trials and future studies
A. Tactics trial and space trial is there. Tactics trial showed significant improvement in PFS if combined with TACE as compared to TACE alone.

Q. Patient BCLC stage c on sorafenib having hand and foot syndrome.should we continue or v hav other options available in india
A. If there is hand foot syndrome it has seen that these patients are more responsive to sorafenib howvever if side effects are severe we can give lenvatinib 8mg/day as a first line therapy which is available in India.

Q. What is the role of genetic profiling of patient with HCC and its role in early diagnosis and treatment ? and the different methods
A. Genetic profiling and molecular targeting can help in future to target specific gene and development of therapeutic vaccine.

Q. Won't you suggest PET to R/O distant mets before undertaking major resection/Transplant?
A. HCC is poorly FDG avid however metastatic lesion show FDG avidity. Most common site of metstasis is lungs, bone and lymphnodes which can be seen with bone scan and ct chest

Q. TARE vs TACE for HCC with PVT?
A. TARE is preferable.

Q. How common is immune related side effects with immune checkpoint inhibitors?
A. Autoimmune thyroiditis, skin toxicity and colitis are reported in around 10% of cases.

Q. If a patient with CTP C with HCC 4 cm with good PS without MVI/EHS has a willing donor, will you attempt LRT before LDLT or go for a LDLT directly or deny the LT
A. CTP C pts will have poor liver reserve which is not suitable for locoregional therapies however if performance status is good LT should be suggested as there are other criterias like UCSF, up to 7 which enrol these patients in transplant list.

Q. TDF or ETV which drug better in prevention of HCC
A. Don't differ significantly

Q. Anyr of HCC surveillance in patients recovered from recovered alcoholic hepatitis ? If not on abstinence
A. Almost 60 to 80 % of cases of severe alcoholic hepatitis have underlying cirrhosis. If cirrhosis is found, surveillance should be done.

Q. How does coffee decrease the risk of HCC ?
A. Coffee ingradients decreases the oxidative stress, activate MAP kinase and improve liver metabolism.

Q. Sir... Does STATIN use along with NUC or DAAs reduce the chances of HCC development in those with HBV AND HCV patients??
A. a systemic review on statins didn't find any risk reduction in HCC.

Q. Do miriplatin in clinical practice has advantage over cisplation in usage of TACE
A. No advantage over cisplatin

Q. Can TACE be used in pts with small PV branches thrombosis in BCLC B ?
A. Can be used

Q. how TARE can be used even in thrombosis
A. because TARE particles are very small so they don't hamper the blood supply to liver and are super selective to tumor only.

Q. Is there any guideline for use of expanded milan or ucfs over conventional milan?
A. UCSF is found as similar to milan in some studies but it is still not as validated as milan.

Q. What is the best criteria for response assessment in patients of HCC ? mRecist Recist or EASL
A. Neither is recommended over the other.

Q. Availability of second line drugs like Regorafenib in india
A. Available

Q. Maximum no of TACE can be done for hcc?
A. no clear recommendation on it but if there is no response after 2 session it is highly unlike to respond further.

Q. Post tace surveillance and management
A. Repeat CT at 1 month to assess the response

Q. When do we need to screen for extrahepatic spread in HCC ? Evidence ?
A. Before prescribing the treatment and for prognostication.

Q. What about combination treatment like pd1 inhibitors and tyrosine kinase inhibitors from beginning in advanced hcc
A. Not recommended. Needs RCTs on this.

Q. Young male patients with unresectable multifocal large HCC with HBV (non-cirrhotic) detected at time of diagnosis. So all HBV patients should be treated in immune tolerant phase to prevent HCC?
A. According to the guidelines only Asian pts > 40 years, family history of cirrhosis or HCC should be treated .

Q. What are the options for BCLC 3 other than drugs?
A. SBRT, SBRT then TACE if no extra hepatic spread. If extrahepatic spread is there then only Systemic therapies.

Q. Is there role of TACE of only the dominant lesion if 1 or 2 small lesions are present in opposite lobe ?

A. Can be used for dominant lesion for downstaging and then transplant.

Q. Which all immunne checkpoint inhibitors are FDA approved for HCC
A. Nivolumab, Pembrolizumab.

Q. What about patient with 6 cm lesion with child A and good performance status No vascular invasion What will you prefer surgery or transplant
A. TACE then Transplant

Q. HCV associated HCC Whether to start DAA before or after transplant .
A. Can be started before or after transplant. If before transplant started it should not hamper patients listing for LT. Post transplant SVR is equally good

Q. What is Role of GALAD score in early detection of HCC in India sir.?
A. GALAD score is not validated in all group of patients. Only evidence have found in noncirrhotic NASH patients.

Q. IN untreated HBV or HCV cirrhosis with high viral load, can TACE for HCC be done immediately after starting antivirals ? Is there any data on post TACE decompensation due to high viral replication ?
A. Few studies has shown high risk of decompensation if there is high viral load, however these are not a contraindication for TACE.

Q. Role of proton beam RT in HCC treatment algorithm
A. In Japan where TARE is not available they use PBT as a treatment modality in HCC with PVTT.

Q. What is the surveillance criteria after liver transplantation
A. No robust consensus on that but 3 to 6 monthly USG for 2 yr is good enough.

Q. What is the maximum duration sorafenib can be given on HCC
A. No consensus. If there is progressive disease or intolerant to sorafenib then it should be stopped.

Q. Which is better between TARE and SBRT in HCC with portal vein invasion?
A. No difference in overall survival.

Q. Role of irreversible electroporation in management of hcc
A. Newer modality. Further studies are needed with long term data. Mostly patients who are candidate for thermal ablation but have limitation due to one or other reason should be benefitted with IRE.

Q. Can lenvatinib be used in portal vein thrombosis
A. No data on lenvatinib in PVTT.

Q. Should we advise cirrhotic patients to drink coffee everyday ?
A. Yes.