## **Conclusions**

HCC is the commonest primary tumour of the liver. There is increase in the rate of incidence of HCC in the world. HCC is the 3rd common cancer in males. It is the 5th common cancer in females. There is increase in the incidence of HCC in Egypt. This may be due to increased exposure to risk factors e.g HBV , HCV and liver cirrhosis.

The risk factors to HCC are multiple and may include:liver cirrhosis, chronic HBV infection, chronic HCV infection, hereditary haemochromatosis, hereditary tyrosinemia, autoimmune hepatitis, nonalcoholic steatohepatitis and other risk factors.

There are changes which occur in the liver to develop HCC e.g increased proliferation of hepatocytes , formation of monoclonal population of hepatocytes , remodeling of liver matrix , neoangiogenesis and aberrant regulation of proliferation of hepatocytes.

The gross pathology of HCC may be nodular, massive or diffuse. The histologic type of HCC may be well differentiated, moderately differentiated, poorly differentiated or undifferentiated. There may be variation in the histologic types of pseudoglandular/acinar pattern, compact/solid pattern, HCC e.g fibrolamellar pattern or schirrous pattern. There may be variations in the cytologic types of HCC e.g giant cell HCC, clear cell HCC, sarcomatoid HCC. There may be intracytoplasmic inclusions in HCC e.g reticular hyalines, globular hyalines or pale bodies. There may be extrahepatic metastasis due to HCC e.g bone and lungs. There may be unusual growth due to HCC e.g pedunculated growth, intrabile duct growth or vascular growth.

HCC is common in patients > 39 years old. The common symptoms due to HCC may be abdominal pain, malaise, anorexia, nausea, vomiting, variceal bleeding, loss of weight and jaundice. The common signs due to HCC may be hepatomegaly, jaundice, hepatic bruit, ascitis, splenomegaly. There may be spontaneous bacterial peritonitis, metastasis, paraneoplastic syndrome e.g diarrhea, erythrocytosis, hypoglycemia or carcinoid syndrome.

The screening to HCC could be done by s.AFP alone or imaging methods alone e.g US , CT , MRI or a combination of s. AFP and imaging methods.

The diagnosis of HCC should include the following:

- 1-serum HCC tumour markers: e.g s.AFP or s. DCP . s. AFP > 400 ng/ml is diagnostic of HCC.
- 2-Imaging methods:e.g US, CT, MRI, scintigraphy or angiographic imaging. US can be used in combination with s. AFP to screen to HCC. These imaging methods can detect vascular invasion of HCC. 3-PANB.
- 4-Laparscopy and laparscopic US.
- 5-Laparotomy and intraoperative US.
- 6-Assesment of the liver functions:It could be done by the Child-Pugh scoring system or by the Model for End Stage Liver Disease scoring system.
- 7-Dynamic measurement of liver functions.
- 8-Measurement of portal pressure and the hepatic blood flow.

There are diferrent staging systems to HCC e.g BCLC , CLIP , Okuda or TNM staging systems.

There are different treatment options to HCC e.g tumour resection , liver transplantation , PEI , RFA , TACE , antineoplastic drugs or symptomatic treatment.

Prognosis of HCC depends on tumour aetiology, tumour pathology, tomour biology, host factors or treatment factors e.g HCC in females has a good prognosis than HCC in males.

Prevention of HCC could be done at 3 levels:

- 1-Primary prevention:Prevention and treatment of the cause of HCC e.g prevention and treatment of HBV infection.
- 2-Secondary prevention:e.g by polyprenoic acid, cholorophyllin or oltipraz.
- 3-Tertiary prevention:e.g by glycyrrhizin.