

## Review Article

# Cardiac and Hepatic Cancers with their Treatment Strategies

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### Abstract

This review aims to sketch current scenario of cardiac and hepatic cancers, which deals with the treatment strategies of these two cancers.

**Keywords:** Cancer; Heart; Liver; Treatment Strategy

### Introduction

The extremely rare form cancer is Heart Cancer (HC) or Cardiac Cancer (CC). HC is divided into primary and secondary tumors of the heart. Metastatic tumors occur twenty times more frequently than the primary HC [1]. HCs usually may develop from any part of the heart. Benign myxomas are the most common HC. Angiosarcomas is a common malignant HC in adults, develops in the right upper chamber of the heart (atrium). It actually originates from the cells that form the lining of blood vessels. Cardiac rhabdomyosarcoma (origin: muscle cells) is the second most common primary HC in adults, while first to the children. It can develop anywhere in the heart, but myocardium involvement is crucial. Few other less common types of primary HCs are cardiac angiosarcoma or cardiac sarcoma, mesotheliomas, fibrosarcomas, fibrous histiocytomas and schwannomas. Symptoms of HCs include - common: chest pain or pressure, cough (pink, frothy sputum), fatigue, fever, irregular heart rhythm (arrhythmia), shortness of breath, swelling in the feet and ankles, unexpected weight gain or loss, weakness (loss of strength), widening and thickening of the fingertips (clubbing) and serious: bluish coloration of the lips or fingernails, change in level of consciousness or alertness, change in mental status or sudden behavior change (such as confusion, delirium, lethargy, hallucinations and delusions), chest pain, chest tightness, chest pressure, palpitations, irregular heart rhythm, coughing-up blood (hemoptysis), less or even no urine production, paralysis or inability to move a body part, rapid heart rate (tachycardia), respiratory or breathing problems (e.g.- shortness of breath, difficulty breathing, labored breathing, wheezing, not breathing and choking).

Like other malignancies, HCs can spread to other distant organs. Otherwise, tumor fragments can break free and enter into the circulation. Those are responsible to cause stroke, oxygen deprivation (anti-angiogenic effect) to cells of vital organs and limbs, and premature death. Most frequent complications of HCs include: arrhythmias (abnormal heart rhythms), heart failure, cancer metastasis, stroke and tumor emboli.

A major site for drug metabolism and clearance, liver plays a vital role in drug disposition; consequently, alterations in liver function cause alterations in drug disposition. The liver plays a large role in first-pass metabolism because the small intestine, where most orally administered medications are absorbed, empties into the hepatic portal circulation [2]. Liver Cancer (LC) is also known as hepatic cancer. LC is the sixth most frequent cancer, and the second leading cause of cancer death. Cholangiocarcinoma is associated with sweating, jaundice, abdominal pain, weight loss and liver enlargement, while Hepatocellular Carcinoma (HCC) is associated with abdominal mass and pain, emesis, anemia, back pain, jaundice, itching, weight loss and fever. Most frequent cause of LC is cirrhosis due to hepatitis B, C and D, or alcohol. Genetic and epigenetic changes may take place by the hepatitis viruses. Viral infections and/or alcohol consumption are the leading causes of liver cirrhosis [3-5]. Aflatoxins cause LC, especially, HCC through genetic mutation of a gene required for the prevention of cancer via p53 [6]. Precancerous lesions, obesity (steatohepatitis), smoking and diabetes (HCC), primary sclerosing cholangitis and liver fluke (cholangiocarcinoma) are the most common causes of LCs in adults [7-11]. LCs in children, mainly caused by Beckwith-Wiedemann Syndrome, familial adenomatous polyposis, low birth weight and Trisomy 18 related hepatoblastoma, while progressive familial intrahepatic cholestasis is associated with HCC [12,13].

Medications that bind to plasma proteins can have an increased free plasma concentration in chronic liver disease [14]. However, drug metabolism and elimination depend on some factors such as intrinsic drug clearance, hepatic blood flow, and the drug extraction ratio [14-16]. Most of the antiepileptic drugs undergo metabolism through CYP2C9, CYP2C19, and CYP3A4 can cause significant alterations in the pharmacokinetics of these medications, thus affect liver functions [17-19]. Analgesics such as acetaminophen, alcohol, morphine and so on are evident to cause serious hepatotoxicity [20-23]. The sedatives, lorazepam, midazolam and dexmedetomidine also impair hepatic functions [24,25]. Moreover, drugs acting on CVS such as vassopressors,  $\beta$ -blockers and antiarrhythmics [26,27]; lung e. g. - sildenafil which undergoes metabolism via CYP3A4 and CYP2C9 may cause liver dysfunction [28,29]. Proton pump inhibitors [30], antiemetics [31], renal dysfunction, thromboembolism [32] and thrombocytopenia [33] are also linked to liver diseases. Infectious diseases, MDR pathogens and a number of antimicrobials are the leading causes of liver carcinomas [34].

The HCC accounts approximately 75% of all primary LCs. HCC occurs in liver cells known as hepatocytes. LCs can form within the liver, such as the bile duct (cholangiocarcinoma and cholangiocellular cystadenocarcinoma), blood vessels (angiosarcoma and hemangioendothelioma, embryonal sarcoma and fibrosarcoma), muscles (leiomyosarcoma and rhabdomyosarcoma) and immune cells. Carcinosarcomas, teratomas, yolk sac tumours, carcinoid tumours and lymphomas are known as some less common LCs [35,36].

Besides debate, the dynamic contrast-enhanced ultrasonography relative to DCE-CT, CT and MR imaging in liver lesion detection are still used by a number of countries [37-39]. Endoscopic Retrograde Cholangiopancreatography (ERCP) and Magnetic Resonance Cholangiopancreatography (MRCP) are also used in LCs detection [40]. The aim of this review is to note down current circumstances on heart and liver cancers.

## Treatment Strategy of HC

Dietary counseling is the primary concern in healthy heart function. Surgery, physical therapy, heart transplantation, radiation therapy, chemotherapy and palliative care are the effective treatment in HCs. Additionally, complementary and/or alternative complementary treatments with traditional medical supplements and clinical medicines are also useful in HCs. However, extensive care should be taken for complementary treatments, especially in nutritional supplements or homeopathic (nonprescription) remedies. In this context, acupuncture, massage therapy, nutritional dietary supplements, herbal remedies, tea beverages, similar products and yoga may be well thought-out.

## Treatment Strategy of LC

Vaccination against hepatitis and reducing alcohol abuse, aflatoxin exposure (e.g.- by chlorophyllin), obesity, and diabe-

tes can reduce the rates of LCs. Diet control in hemochromatosis could decrease the risk of iron overload, decreasing the risk of cancer [41]. Cytotoxic drugs such as doxorubicin or cisplatin with lipiodol are used in a procedure known as transarterial chemoembolization in HCC. However, these are not effective treatment in HCC. Surgical resection and liver transplantation are often the treatment of choice for non-cirrhotic and HCC, respectively. Percutaneous ablation (ethanol or acetic acid, or producing extremes of temperature using radio frequency ablation, microwaves, lasers or cryotherapy) is the only non-surgical treatment that can offer a cure of LCs including HCC. However, the liver is not tolerated to ionizing radiations; therefore, radiotherapy is not a good idea in HCC [42-45]. Liver transplant, chemo- and/or radiation, radio frequency ablation, transarterial chemoembolization are frequently used in cholangiocarcinoma [13,46]. On the other hand, surgical resection or liver transplant and chemotherapy are the treatment in hepatoblastoma [47,48].

## References

1. Moynihan TJ (2015) Heart cancer: Is there such a thing? *MayoClinic.com* 6: 1.
2. Pang KS (2003) Modeling of intestinal drug absorption: roles of transporters and metabolic enzymes (for the Gillette Review Series). *Drug Metab Dispos* 31: 1507-1519.
3. Fattovich G, Stroffolini T, Zagni I, Donato F (2004) Hepatocellular carcinoma in cirrhosis: incidence and risk factors. *Gastroenterol* 127: S35-S50.
4. GBD (2013) Mortality and Causes of Death, Collaborators. Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 385: 117-171.
5. Arzumanyan A, Reis HM, Feitelson MA (2013) Pathogenic mechanisms in HBV- and HCV-associated hepatocellular carcinoma. *Nat Rev Cancer* 13: 123-135.
6. Kensler TW, Roebuck BD, Wogan GN, Groopman JD (2011) Aflatoxin: a 50-year odyssey of mechanistic and translational toxicology. *Toxicol Sci: Official J Soc Toxicol* 120: S28-S48.
7. Chuang SC, La Vecchia C, Boffetta P (2009) Liver cancer: descriptive epidemiology and risk factors other than HBV and HCV infection. *Cancer Lett* 286: 9-14.
8. Jemal A, Bray F, Center MM, Ferlay J, Ward E, Forman D (2011) Global cancer statistics. *CA: a cancer journal for clinicians* 61: 69-90.
9. National Cancer Institute (NCI) (2013) General Information About Adult Primary Liver Cancer. 13 January 2013.
10. Di Tommaso L, Sangiovanni A, Borzio M, Park YN, Farinati F, et al. (2013) Advanced precancerous lesions in the liver. Best practice & research. *Clin Gastroenterol* 27: 269-284.
11. Razumilava N, Gores GJ (2013) Classification, diagnosis, and management of cholangiocarcinoma. *Clin Gastroenterol Hepatol: Official Clin Pract J Am Gastroenterol Assoc* 11: 13-21.
12. Davit-Spraul A, Gonzales E, Baussan C, Jacquemin E (2009) Progressive familial intrahepatic cholestasis. *Orphanet J Crare Dis* 4: 1.

13. Spector LG, Birch J (2012) The epidemiology of hepatoblastoma. *Pediatric Blood Cancer* 59: 776-779.
14. Verbeeck RK, Horsmans Y (1998) Effect of hepatic insufficiency on pharmacokinetics and drug dosing. *Pharm World Sci* 20: 183-192.
15. Kim JW, Phongsamran PV (2009) Drug-induced liver disease and drug use considerations in liver disease. *J Pharm Pract* 22: 278-289.
16. Lin S, Smith BS (2010) Drug dosing considerations for the critically ill patient with liver disease. *Critic Care Nurs Clin North Am* 22: 335-340.
17. Brockmoller J, Thomsen T, Wittstock M, Coupez R, Lochs H, et al. (2005) Pharmacokinetics of levetiracetam in patients with moderate to severe liver cirrhosis (Child-Pugh classes A, B, and C): characterization by dynamic liver function tests. *Clin Pharmacol Ther* 77: 529-541.
18. Asconape JJ (2014) Use of antiepileptic drugs in hepatic and renal disease. *Handb Clin Neurol* 119: 417-432.
19. Anderson GD, Hakimian S (2014) Pharmacokinetic of antiepileptic drugs in patients with hepatic or renal impairment. *Clin Pharmacokinet* 53: 29-49.
20. Yogaratnam D, Miller MA, Smith BS (2005) The effects of liver and renal dysfunction on the pharmacokinetics of sedatives and analgesics in the critically ill patient. *Critic Care Nurs Clin North Am* 17: 245-250.
21. Bosilkovska M, Walder B, Besson M, et al. (2012) Analgesics in patients with hepatic impairment: pharmacology and clinical implications. *Drugs* 72: 1645-1669.
22. Dwyer JP, Jayasekera C, Nicoll A (2014) Analgesia for the cirrhotic patient: a literature review and recommendations. *J Gastroenterol Hepatol* 29: 1356-1360.
23. Imani F, Motavaf M, Safari S, et al. (2014) The therapeutic use of analgesics in patients with liver cirrhosis: a literature review and evidence-based recommendations. *Hepat Mon* 14: e23539.
24. Hughes CG, McGrane S, Pandharipande PP (2012) Sedation in the intensive care setting. *Clin Pharmacol* 4: 53-63.
25. Barr J, Fraser GL, Puntillo K, Ely EW, Gélinas C, et al (2013) Clinical practice guidelines for the management of pain, agitation, and delirium in adult patients in the intensive care unit. *Critic Care Med* 41: 263-306.
26. Klotz U (2007) Antiarrhythmics: elimination and dosage considerations in hepatic impairment. *Clin Pharmacokinet* 46: 985-996.
27. Canabal JM, Kramer DJ (2008) Management of sepsis in patients with liver failure. *Curr Opin Critic Care* 14: 189-197.
28. Revatio\_ [package insert]. New York, NY: Pfizer Labs; 2015.
29. Viagra\_ [package insert]. New York, NY: Pfizer Labs; 2015.
30. Nexium\_ [package insert]. Wilmington, DE: AstraZeneca Pharmaceuticals LP; 2014.
31. Zofran\_ [package insert]. Research Triangle Park, NC: GlaxoSmith-Kline; 2014.
32. Wu H, Nguyen GC (2010) Liver cirrhosis is associated with venous thromboembolism among hospitalized patients in a nationwide US study. *Clin Gastroenterol Hepatol* 8: 800-805.
33. Swan SK, Hursting MJ (2000) The pharmacokinetics and pharmacodynamics of argatroban: effects of age, gender, and hepatic or renal dysfunction. *Pharmacother* 20: 318-329.
34. Fernandez J, Acevedo J, Castro M, Garcia O, de Lope CR, et al (2012) Prevalence and risk factors of infections by multiresistant bacteria in cirrhosis: a prospective study. *Hepatol* 55: 1551-1561.
35. Ahmed AI, Lobo DN (2009) Malignant tumours of the liver. *Surgery (Oxford)* 27: 30-37.
36. Khan SA, Davidson BR, Goldin RD, Heaton N, Karani J, et al. (2012) Guidelines for the diagnosis and treatment of cholangiocarcinoma: an update. *Gut* 61: 1657-1669.
37. Bruix J, Sherman M (2005) Management of hepatocellular carcinoma. *Hepatol* 42: 1208-1236.
38. Kudo M, Izumi N, Kokudo N, Matsui O, Sakamoto M, et al. (2011) Management of hepatocellular carcinoma in Japan: Consensus-based clinical practice guidelines proposed by the Japan Society of Hepatology (JSH) 2010 updated version. *Dig Dis* 29: 339-364.
39. Bolondi L (2013) The appropriate allocation of CEUS in the diagnostic algorithm of liver lesions: a debated issue. *Ultrasound Med Biol* 39: 183-185.
40. Ariff B, Lloyd CR, Khan S, Shariff M, Thillainayagam AV, et al. (2009) Imaging of liver cancer. *World J Gastroenterol: WJG* 15: 1289-1300.
41. Hoshida Y, Fuchs BC, Tanabe KK (2012) Prevention of hepatocellular carcinoma: potential targets, experimental models, and clinical challenges. *Curr Cancer Drug Targets* 12: 1129-1159.
42. Bruix J, Sherman M, American Association for the Study of Liver, Diseases (2011) Management of hepatocellular carcinoma: an update. *Hepatol (Baltimore, Md.)* 53: 1020-1022.
43. Feng M, Ben-Josef E (2011) Radiation therapy for hepatocellular carcinoma. *Seminars Radiat Oncol* 21(4): 271-277.
44. de Lope CR, Tremosini S, Forner A, Reig M, Bruix J (2012) Management of HCC. *J Hepatol* 56: S75-87.
45. Wang ZG, Zhang GF, Wu JC, Jia MK (2013) Adjuvant therapy for hepatocellular carcinoma: Current situation and prospect. *Drug Discoveries Therapeutics* 7: 137-143.
46. Kuhlmann JB, Blum HE (2013) Locoregional therapy for cholangiocarcinoma. *Curr Opin Gastroenterol* 29: 324-328.
47. Meyers RL, Czauderna P, Otte JB (2012) Surgical treatment of hepatoblastoma. *Pediatric Blood Cancer* 59: 800-808.
48. Perilongo G, Malogolowkin M, Feusner J (2012) Hepatoblastoma clinical research: lessons learned and future challenges. *Pediatric Blood Cancer* 59: 818-821