CASE REPORT: TO REPORT THE RARE PRESENTATION OF DVT WITH HEPATOMA IN HCV +VE CASE OF CLD.

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ABSTRACT

BACKGROUND: Hepatoma is a common liver tumor in viral hepatitis C with and without complications. **AIM:** to report the rare presentation of DVT with CLD and hepatoma. **SETTING:** PMCH Nawabshah. **TYPE OF STUDY:** case report. **DURATION:** 10-08-2019 to 30-08-2019. **METHOD:** a single rare case of HCV and hepatoma with DVT was admitted in department of medicine, admitted and investigated. After diagnosis it is reported. **RESULT:** a 55 years old male known case of HCV and hepatoma presented here with swelling of right lower limb, color Doppler studies had confirmed the extensive venous thrombus in leg, with complete occlusion of deep veins of right lower limb. D-dimers were >600ngm. **CONCLUSION:** DVT is not common in HCV and hepatoma associated CLD cases. It is rare one. Early diagnosis and multidisciplinary management can be a promising step for good prognosis. **KEY WORDS:** DVT, CLD, Hepatoma, HCV

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INTRODUCTION

Subjects with malignancy are susceptible to to venous thromboembolic difficulties (Venous Thomboembolism), possibly owing to overexpression of tissue factor, increased platelet stimulation by malignancy promoting proteins, & above excretion of cytokines ¹. Extra reasons such as chemotherapy, hormone central catheter treatment. & venous employment could too rise the hazard of Venous Thomboembolism². Cancer-related surgery is also associated with a higher risk of Venous Thomboembolism than other surgery ^{3,4}. Not surprisingly, Ca Liver (hepatocellular carcinoma) is moreover related with an bigger threat of Venous thromboembolism, through the PV being the furthermost shared location for thrombosis (Portal Vein Thrombus), through an occurrence of 20-40% reported.⁵ Because this cancer is most often a complication of cancer, it is necessary to consider the circumstances under which Ca Liver develops, in order to recognize how the occurrence of Hepatocellular Ca disrupts the hemostatic balance.⁶ The prevalence of cirrhosis in HCC patients is estimated to be 85-95% ⁷. A balanced and unstable hemostasis of cirrhosis can easily fall in the direction of thrombotic complications depending on the superposition, including HCC⁸. In addition, patients with cirrhosis can develop non-visceral thrombosis, and patients with cirrhosis do not have self-anticoagulant effects as previously thought, but are actually more prone to thrombotic complications than patients with non-cirrhosis. It is becoming more and clearer ⁹. HCC can develop in the liver with non-cirrhosis. According to a very recent review by Kulik L. et

al.¹⁰ The incidence of HCC without prior cirrhosis is abnormal, especially in about 15% of HBV-related cases. HBV is known to be directly involved in liver mutagenesis. In addition, non-alcoholic fatty liver disease (NAFLD) and metabolic syndrome accounted for the majority of new cases of HCC without advanced fibrosis or cirrhosis in the United States ¹¹.

CASE HISTORY: 64 years old male salesman by occupation resident of N-shah come through emergency with complain of abdominal pain > 3 months. Mass in right hypochondrium for 01 month. Bleeding in vomitus and stool > 1 week. Painful ful swelling of right lower limb for 3 Days.

Co-morbidity – having hepatitis C for 3 years. HOPC: According to patient he was in usual state of health 3 months back then he develop abdominal pain which was located in right hypochondrium gradual in onset ,mild to moderate in intensity ,dull aching in character ,not radiating ,no relation with meal and posture .pain is related by fever low grade , , loss of appetite and weight about 5 kg of same duration, is blood in other complain vomitus (hematemesis) which is fresh red in color, small in quantity, 2-3 time in frequency and associated melena. No history of jaundice ,loss of consciousness, SOB, cough, no history of abdominal distension ,swelling of feet, no history of petechial, purpura, bruises, bleeding gums , regarding hepatitis c which was diagnostic via HCV-Ab 3 month back for that he is not taking treatment, regarding risk factor there is no history of blood transfusion ,needle pricking and tattooing ,i/v drug abuser , alcohol, history of shaving by salon. Functional class =

patient is unable to perform his daily routine activities, he is on bed more than half of the day, after need help to look yourself. Past medical history = history of pulmonary TB 6 years back for that he taking ATT 3 tab /day for 9 months .- Past surgical history = not significant . Personal history = Appetite decreased. Bladder hobbit -normal. Bowel hobbits - increase in frequency 2-3 episode history of melena. Sleep –Disturb. Addiction history = cigarette smoker 2-3 cigarettes /day. Family history = not significant. Family screening for hep B and C is not done. Socioeconomic history = he belongs to middle class family having a cemented house ,well ventilated ,using hand pump water for drinking ,no pets and domestic animal at home surrounding is satisfactory .home Vaccination= Nil. Travel history = Nil. Drug history = taking symptomatic treatment he don't remember names of drugs. Allergy history =no any history of allergic disorder.

GERNERAL PHYSICAL EXAMINATION: old age male normal height, thin, looking ill lying uncomfortable on bed .VITALS BP= 110/70 mmhg. Pulse= 70b/min. Temp= 98F. R/R=18 br/min. SUBVITALS. Anemia+. Jaundice+ CLubbing+, Edema +, Cyanosis-, JVP-, ABDOMEN EXAMINATION; on mildly distended, move with inspection: respiration, umbilical is everted. Veins are prominent, on superficial and deep palpation : abdomen is hard tender mass is palpable at Right and left hypochondrium. On visceral palpation: Liver is palpable, 8cm below Right costal margin in midclavicular route. Edge is sharp, exterior is irregular, tender, stable in stability, & superior margin of hepatic dullness is in right 5^{th} I/C (Intercostal space) in mid-clavicular line on right side. There is audible hepatic bruit. Spleen is enlarge 4 cm from costal margin left side in the anterior axillary line in the direction of iliac fossa (right) 9cm starting costal border in left midclavicualr route. Hard in consistency, notch of spleen is present, on percussion there is dullness over spleen which is continuous up to left lower part of chest. Other organ are not palpable SD +ve .FT –ve. **CVS** = un-remarkable .CHEST= un-remarkable. CNS= remarkable.

Routine Investigation. CBC, UCE, LFT, PT, APTT, Viral Markers, ICT -Malaria, Typhidot, Chest X-Rays, U/S of Whole Abdomen. Investigation. Specific Alpha-Fetoprotein.990.4iu and Triphasic CT-scan of abdomen with contrast. Colour Dropler Of Right Lower Limb: Extensive Deep Vein Thrombosis of Right Leg. **CT-SCAN** ABDOMEN FINDING: Liver is enlarged showing irregular margin with altered left to right lobe ratio .portal vein is dilated measure 1.6cm .right branch of portal vein is not clearly appreciated either could be due to portal vein thrombus or infiltrated by mass, main portal vein and left branch appear intact, spleen is bulky, varices are splenic hilum, portahepatis, perigastric and peripancreatic region .these finding are due to chronic liver disease .There is

large arterial based heterogeneous lesion noted in almost completely occupying in the right lobe of liver. It measure 17.9x12.2x24.3cm in APXTSXCC DIMENSION most likelv multicentric hepatoma. Conclusion: Above described findings are due to CLD with multicentric hepatoma. Child-Pugh Score Hepatic Encephalopathy +2, Ascites +2Bilirubin (Total) +2, PT +2 Albumin +1. Total=9 **Meld Score**: Meld=3.78x(S-Bilirubin Mg/Dl) +11.2x (INR) +9.57(S-Creatinine Mg/Dl)

Meld=3.78x2.7+11.2x1.48+9.57x0.5+6.43= 37.997.

Diagnosis: Hepatocellular Carcinoma (Hepatoma)

TREATMENT PLAN IS?

- ➢ Resection.
- Radiofrequency Ablation.
- Percutaneous Ethanol Injection.
- ➢ Liver Transplant,
- Trans arterial Chemo-Embolization
- ➢ Sorafenib.
- Best Supportive Care.

DISCUSSION

It is well known that subjects having malignancy are disposed to venous thomboembolism (venous thromboembolic) problems. Therefore, it is not astonishing that subjects with Ca Liver are at major threat of venous thromboembolism & portal vein is the utmost shared dwelling for Portal Vein Thrombus. Though, subjects with Ca Liver are unique in that together malignancy & liver cirrhosis could unsettle the hemostatic equilibrium in the direction of a thrombuspromoting condition. Since no Ca Liver -linked hypercoagulation has been elucidated, the purpose of this case presentation is to review the present formal of information nearby the epidemiology & etiology of non-cancerous thrombotic problems in subjects through cirrhosis of liver & Ca Liver The risk of developing together Portal Vein Thrombus and non-visceral Venous Thomboembolism is in elevation, suggesting that in cooperation native and general causes may support the progress of definite location thrombosis. Current revisions suggest numerous, & frequently consistent, tools by which Ca Liver can tilt the hemostatic equilibrium of liver cirrhosis in the direction of hypercoagulation.. Mechanisms defined comprise augmented fibrinogen levels thrombocytosis, polymerization, & discharge of extracellular vesicles expressing tissue factor. Presently, here are no exact guiding principle for the use of thrombus prophylaxis in this sole populace. Prospective studies are urgently needed to investigate which subjects take the uppermost thrombus-promoting outline & consequently advantage from initial thrombosis prophylaxis. ¹² Liver cancer is at increased risk of developing thrombotic complications other than internal organs. In particular, the incidence of Venous Thomboembolism complications was examined in three large studies. Levitan N in 1999 et al. ¹³ examined 22,938 liver cancer

subjects via billing statistics of Medicare & described a frequency of 69 venous

thomboembolisms per 10,000 subjects.

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great venous thomboembolism hazard (lymphoma, kidney, pancreas, brain, stomach, subjects with ovary,) & low venous thomboembolism risk (esophagus, breast. bladder, uterus head & neck,) /10,000 subjects. The incidence was 16-50 people per per. A succeeding revision by Wun and White in 2009 confirmed an average risk of liver cancer, with a 1.7% frequency of Venous Thomboembolism in 01 year of conclusion of Ca Liver, equivalent to ca lung.¹⁴. A cohort study populace centered thru Cronin Fenton et al. 1.1% cumulative incidence in 2010, was reported in 550 subjects with liver cancer during a one-year follow-up period ¹⁵. The concurrency rate in the overall populace was 0.4%. Remarkably, the incidence of Venous Thomboembolism was at peak in subjects with hepatic, pulmonary, pancreatic, ovarian, & head cancers. Additionally, in the identical revision, the writers described a great frequency of Venous Thomboembolism hospital admissions in subjects with Ca liver (20.4 per 1000 person-years (9.2-45.3)). Inappropriately, those researches did not differentiate among Ca Liver subjects with liver cirrhosis & without cirrhosis of liver. The increased hospital admission frequency might be owing to coof cirrhosis of occurrence liver. Subjects with liver cirrhosis are at increased hazard of emerging non-visceral thrombotic difficulties themselves ¹⁶. Or pulmonary embolism (PE) occurred retroactively. 0.8-7% of subjects hospitalized with cirrhosis develop Venous Thomboembolism^{16,17,18,19,20,21,22,23,}

Venous Thomboembolism problems than acute subjects deprived of hepatic illness was evaluated in a case-control study that did not show. ^{16,24}, and above all, long-term INR did not affect this risk ²⁵. In two groundbreaking studies, low albumin levels are self-determining forecaster of Venous Thombo-embolism in multivariate investigation & may reflect small heights of anticoagulants found endogenously usually originate in coagulopathy with liver cirrhosis. ^{16, 24}. On the other hand, the old-fashioned indicators liver disease with coagulopathy, (eg, platelet count INR), does not forecast and Venous Thomboembolism ²⁴ & not should be measured in medical exercise.

COCLUSION

Our case study concluded that subjects with Chronic Liver Diseases and malignancy should be routinely screened for the extra hepatic manifestation of complications of hepatomas

ETHICS APPROVAL: The ERC gave ethical review approval CONSENT TO PARTICIPATE: written and verbal consent was

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