Case Report

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When Heart Kills Liver: Nutmeg Liver: Case Report

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Abstract

Congestive hepatopathy includes a spectrum of hepatic derangements that occur in the settings of Right heart failure. If there is subsequent hepatic fibrosis the term cardiac cirrhosis may be used. The timely diagnosis of a cardiac etiology of liver dysfunction is important because such dysfunction is potentially reversible if the underlying cardiac disease is treated before the development of frank cirrhosis. Below, we present a case of 18 years old adolescent girl who was incidentally found to have Atrial Septal defect. Abdominal USG revealed Nutmeg Liver. In this case report the literature will be discussed on cardiac events resulting in Nut Meg liver and its management.

Introduction: Congestive hepatopathy is also known as Nutmeg liver and chronic passive congestion of the liver, which is an liver dysfunction due to venous congestion, usually caused by congestive heart failure, The gross pathological appearance of a liver with Nutmeg is Speckled like a grated nut meg kernel [6]. Treatment is directed to remove the cause. So, therapy aimed at improving Right heart function. True nutmeg liver is usually second to left sided heart failure causing congestive right heart failure and the treatment options are limited [12].

Case Report: A 18 years old adolescent girl with a known atrial septal defect, cystichygroma in the left neck, presented to the cardiology department complaining of shortness of breath (grade IV), non productive cough, orthopnea, fatigue. On physical examination the findings were Tachypnea, cold extremities and bibasilar crackles on pulmonary auscultation; An abdominal ultra sound was requested and the findings were congestive hepatopathy, splenomegaly minimal left pleural effusion and moderate volume of ascities. X-ray showed cardiomegaly obliterating left CP angle, patchy air space opacities, right mid zone and lower zone pulmonary edema changes. 2D Echo cardiogram showed cyanotic heart disease, Large oss with atrial septal defect with predominantly left to right shunt, dilated right atria, right ventricle& pulmonary artery; severe tricuspid regurgitation with moderate pulmonary artery hypertension, Right ventricular dysfunction with moderate atrial regurgitation; mild pulmonary edema, global hypokinesia of left ventricle, mild left ventricle systolic dysfunction.

Conclusion: The study highlight cardiac cause should be thought for differential diagnosis when patient presents with liver cirrhosis; the incidence of patients with Nutmeg liver are presenting in general practice.

Keywords: Congestive Hepatopathy; Right Heat Failure; Atrial Septal Defect; Nut Meg Liver; Congestive Heart Failure; Liver Dysfunction; Cirrhosis of Liver.

Introduction

Congestive hepatopathy is also known as Nutmeg liver and chronic passive congestion of the liver, which is an liver dysfunction due to venous congestion, usually caused by congestive heart failure, The gross pathological appearance of a liver with Nutmeg is Speckled like a grated nut meg kernel; The dart spots represent the dilated and congestive hepatic venules and small hepatic veins; long standing hepatic congestion can lead to fibrosis ,if the cause is right heart failure it is called cardiac cirrhosis [7,8]. USG, Doppler studies of the portal and hepatic arteries and veins; ECG and Echo cardiogram are useful tools to detect hepatic congestion and assess fibrosis; MRI MR elastrography are understudy [5]. In ascities cases paracentesis should be done and high protien in ascitic fluid reflects rupture of hepatic lymphatic system. A marked elevated serum N-terminal pro BNP level distinguished ascities due to heart failure from ascities related to cirrhosis. Improvement

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in liver biochemical tests with treatment of the underlying cardiac conditions provides support for diagnosis [6]. Treatment is directed to remove the cause. So, therapy aimed at improving Right heart function. True nutmeg liver is usually second to left sided heart failure causing congestive right heart failure and the treatment options are limited [12].

Case Report

A 18 years old adolescent girl with a known atrial septal defect, cystichygroma in the left neck, presented to the cardiology department complaining of shortness of breath (grade IV), non productive cough, orthopnea, fatigue. On physical examination the findings were Tachypnea, cold extremities and bibasilar crackles on pulmonary auscultation: signs and symptoms of decompensate CHF prompted further evaluation with 2D Echo cardiogram which showed cyanotic heart disease, Large oss with atrial septal defect with predominantly left to right shunt, dilated right atria, right ventricle & pulmonary artery; severe tricuspid regurgitation with moderate pulmonary artery hypertension, Right ventricular dysfunction with moderate atrial regurgitation; mild pulmonary edema, global hypokinesia of left ventricle, mild left ventricle systolic dysfunction. An abdominal ultra sound was requested and the findings were congestive hepatopathy, splenomegaly minimal left pleural effusion and moderate volume of ascities. X-ray showed cardiomegaly obliterating left CP angle, patchy air space opacities, right mid zone and lower zone pulmonary edema changes.

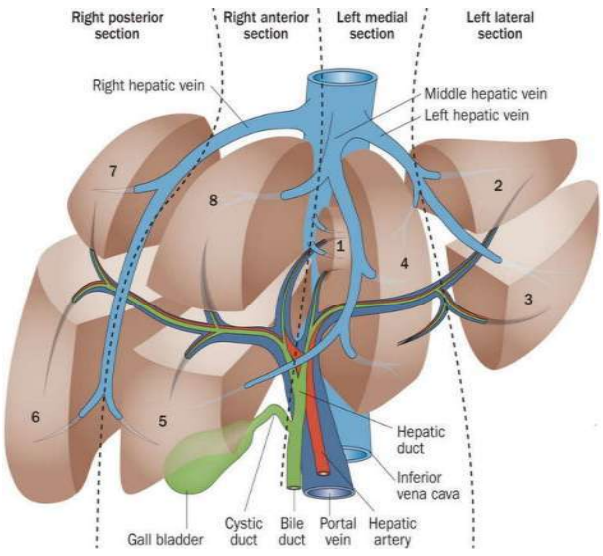

Fig. 1: Physiological Anatomy of Liver

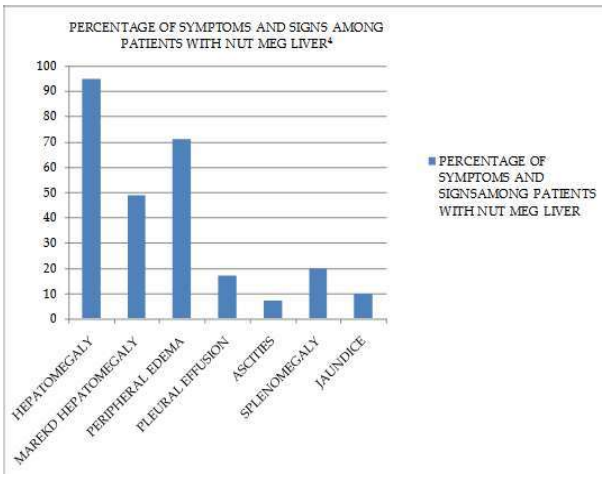

Fig. 2: Percentages of Symptoms and Signs in Nutmeg Liver

Blood test revealed elevated total bilirubin levels (3.9 mg/dl), direct bilirubin 1.7 mg/dl, indirect bilirubin 2.2mg/dl, SGPT 13 U/L, serum albumin 2.9gm/dl, globulin 5.1gm/dl, raised total iron binding capacity 387 µgm/dl, serum iron 42µg/dl, serum ferratin 10.94ng/ml, with Hb-6.0gm/dl, serum sodium level as 129 mmol/lit. Prothrombin concentration - 71.6%, International Normalized Ratio 1.46. The client was diagnosed as Nutmeg liver which is also called as congestive hepatopathy. Accordingly the patient was placed on symptomatic treatment T.frusalac 40 mg, T.lanoxin 0.25mg, T. augmentin 625 mg, T.somfral 40 mg, Syr. Ascoril 5mg, T Motar LC, T Abflo 100 mg. comprehensive Nursing care was carried as per the needs and priorities by using nursing process.

Discussion

Liver is a largest solid organ, weighing approximately 3lb (1500 gms), located in the right upper quadrant, beneath the diaphragm. liver consists of three lobes divided in to eight independent segments, each of which has its own vascular in flow, out flow and biliary drainage, because of this division into self contained units, each can be resected without damaging those remaining.

Right lobe: Anterior (segments V and VIII) and posterior (segments VI and VII).

Left lobe: medial (segment-IV) and Lateral (segments II and III); the left lobe extends across the midline in to the left upper quadrant;

Caudate lobe: (segment-I)

Microscopically the liver consists of functional units called lobules composed of portal triads in

which the bile ducts, hepatocytes and artery are located. The portal triads are then bounded by sinusoids and a central vein. Across section of a classic lobule or acinus is hexagonal. Blood supply derived from both artery and vein. 25% of cardiac output flows through the liver per minute. portal vein (after draining the mesenteric vein and pancreatic an splenic veins) and hepatic artery (off the aorta via the celiac trunk) enter the liver at the porta hepatis or hilum (a horizontal tissue in the liver containing blood and lymph vessels, nerves and the hepatic ducts). 75% is supplied by the portal vein; each segment receives a branch of the portal vein and 25% is supplied by the hepatic artery. Blood from both portal and hepatic artery mixes together in the hepatic sinusoids and then flow through hepatic vessels. Venous drainage begins in the central veins in the center of the lobules; central veins empty in to the hepatic veins, which empty in to inferior vena cava.

The incidence of congestive hepatopathy, significant fibrosis or cardiac cirrhosis ranges between 15%-65% of patients with significant heart failure. By today's accounts cardiac cirrhosis is rare [1].

Pathophysiology alteration as Increased pressure in the sub lobular branches of the hepatic veins causes an engorgement of venous blood, and is most frequently due to chronic cardiac lesions, especially those affecting the right heart, the blood being dammed back in the inferior vena cava and hepatic veins. central regions of the hepatic lobules are red brown and stand out against the non congested, tan colored liver, Centrilobular necrosis occurs [5,6].

Macroscopically, the liver has a pale and spotty appearance in the affected areas, as stasis of the blood causes pericentral hepatocytes to become deoxygenated compared to the relatively better oxygenated peri portal hepatocytes adjacent to the hepatic arterioles. This retardation of the blood also occurs in lung lesions, such as chronic interstitial pneumonia, pleural effusions and intra thoracic tumors [6,8].

Clinical manifestations depend largely up on the primary lesions giving rise to the condition. In addition to the heart or lung symptoms, there will be a sense of fullness and tenderness in the right hypochondric region. Gastro intestinal catarrh is usually present, and vomiting of blood may occur [2]. There is usually more or less jaundice owing to portal obstruction, ascities occur, followed later by generalized edema. The stools are light or clay colored, and urine is colored by bile. On palpation hepatomegaly, tenderness extending several inches below the costal margin of the ribs [3,4].

Term cardiac cirrhosis denotes any type of hepatic fibrosis occurring in cardiac patient. Our case report is in agreement with the previous observations of chronic liver injury due to long term congestive heart failure [9]. Though the incidence of cardiac cirrhosis is low but causes are same like Ischemic heart disease, Cardiomyopathy, Valvular heart disease, Primary lung disease, Pericardial diseases. Incidence of valvular heart disease decreased with increased incidence of, cardiomyopathy as an etiology of cardiac cirrhosis [9,10]. Our case had presented with shortness of breath (grade IV), non productive cough, orthopnea, fatigue. On physical examination the findings were Tachypnea, cold extremities and bibasilar crackles on pulmonary auscultation. On evaluation the cause was cyanotic heart disease, Large oss with atrial septal defect predominantly with left to right shunt, dilated right atria, right ventricle & pulmonary artery; severe tricuspid regurgitation with moderate pulmonary artery hypertension, Right ventricular dysfunction with moderate atrial regurgitation; mild pulmonary edema, global hypokinesia of left ventricle, mild left ventricle systolic dysfunction leading to chronic congestive heart failure. This further leads to passive congestion and relative ischemia due to poor circulation eventually leading to necrosis and fibrosis of liver predominantly of centrilobular region [11,12]. Chronic congestive heart failure established on long history of 6 years for which treatment was consumed of which records were not available. Deranged Liver Function Test with markedly increased bilirubin levels and SGPT. Metabolic and synthetic functions of liver were also compromised evident from decreased serum albumin and deranged PT/INR.

The cornerstone of management of all forms of congestive hepatopathy from asymptomatic mild elevations in hepatic indices to cardiac cirrhosis is targeted toward treating the underlying cardiac function and any triggers accounting for acute decompensation jaundice, hepatic congestion and ascities may respond dramatically to therapy with diuretics; however these drugs should be used with caution to avoid dehydration, hypotension and hepatic ischemia by precipitating zone 3 necrosis [1,2]. It is of vital importance to maintain an adequate cardiac output. Our case was treated symptomatically; Serial large volume paracentesis can relieve symptoms in those with diuretic

refractory tense cardiac ascities, but overtime can lead to protein loss and exacerbate the protein malnutrition commonly seen in those with advanced heart failure [1,3]. Trans jugular intra hepatic Porto systematic shunts or peritoneal venous shunts are contraindicated in this population as they can lead to exacerbation of the underlying heart failure [2]. Cautious use of anticoagulants is advised because patients have a baseline mild increase in PT/INR and are especially sensitive to warfarin and other related compounds. Inpatients refractory to medical therapy who are suitable operative candidates both LVAD implantation have been shown with the failing heart, In patients with established cirrhosis, combined heart and liver transplant is a feasible option. Recently, there has been a report of possible reversal of cardiac cirrhosis with heart transplantation alone, effectively removing the source of the insult. However, such cases are the expectation [2,3].

Classically Nursing Management for Nut Meg liver or cardiac cirrhosis focus on goals such as optimizing remaining liver function, stabilize decompositions and collaborating professionals in health care team. The anticipated patient trajectory with chronic liver failure may plateau prior to decompensation then deteriorate rapidly. Patients may be expected to have needs in the following areas. Development of orthostatic hypotension dictates the need for slow deliberate movements to prevent dizziness and falls; Skin will be dry, there will be an increase in bruising due to reduction of the platelet count and levels of coagulation factors. Our case presented with orthostatic hypotension and dry skin was noted which were handled by nursing interventions such as careful awakening, Elevate the head of the bed (reducing nocturia), Drink two cups of cold water 30 minutes before arising and Shift from supine to an erect position in gradual stages; Treated Anemia, avoided physical exertion, advised not to stain during micturation or defecation, not to consume heavy carbohydrate meal to avoid exacerbation of symptoms.

Ascites may cause early satiety; low zinc levels in liver disease may result in diminished taste or metallic taste; patients develop severe muscle wasting and malnutrition. In our patient minimal ascities and severe muscle wasting was appreciated, so, encouraged to consume small and frequent feeds. Depressed immune system increases the risk of infection; presence of ascities creates the risk of peritonitis. Psycho social issues as chronicity of the situation will have profound impact on the family unit and increase stress; depression can occur in both patient and primary care giver. Ethical issues such as lack of available organs and prolonged hospitalizations increase the risk of sepsis, which prevents transplantation and leads to discussions of with drawl of life support. Discharge planning is vital because recovery periods are short and re hospitalization can be frequent as the patient decompensate. Family and patient needs assistance with home care, rehabilitation, medications, office visits.

Over time, hepatic function typically remains stable and even when cardiac cirrhosis and ascities ensure, patients with congestive hepatopathy rarely develop other features of hepatic insufficiency. Several studies have addressed the prognostic importance of liver function abnormalities as predictors. Batin etal demonstrated that the greatest prognosticators in CHF were AST and total Bilirubin [1].

Conclusion

A patient with ASD with LV dysfunction developing chronic right sided heart failure due to pulmonary hypertension causes passive congestion on hepatic veins leading to relative ischemia and eventually to hepatic necrosis and fibrosis and raised portal hypertension [9]. The study highlight cardiac cause should be thought for differential diagnosis when patient presents with liver cirrhosis; the incidence of patients with Nutmeg liver are presenting in general practice. It is important that nurses understand the common causes, pathophysiological alterations, clinical manifestations and complications of cardiac cirrhosis as well as the complexities of patient management, and should assess the need, prioritize, prevent the complications and restore liver function, stabilize decompensation with collaboration of health team and provide high quality care for the clients with cardiac hepatopathy.

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Conflict of Interest

No conflict of interest

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