ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research

Vol. 16, Issue, 07, pp.29402-29404, July, 2024
DOI: https://doi.org/10.24941/ijcr.47538.07.2024

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

RESEARCH ARTICLE

HOW YOUR HEART CAN DESTROY YOUR HEALTHY LIVER FEATURING CARDIAC CIRRHOSIS: A CASE PRESENTATION

Sumol Ratna[1], Simran Gangwani[2]*, Divyansh Singh[2], Neha Gurjar[2], Arshita Gupta[2], Pragya Dalel[2] and Nishant Sharma[2]

[1]Assistant professor, Department of General Medicine, Noida International Institute of Medical Sciences, Gautam Budh Nagar, Uttar Pradesh, India; [2]UG MBBS, Noida International Institute of Medical Sciences, Gautam Budh Nagar, Uttar Pradesh, India

ARTICLE INFO

Article History:
Received 18th April, 2024
Received in revised form
19th May, 2024
Accepted 25th June, 2024
Published online 30th July, 2024

Key words:
Statelessness, former Refugees, New permanent Resident, Convention, Nationality, and UNHCR.

ABSTRACT

Cardiac cirrhosis represents the spectrum of hepatic disorders occurring secondary to hepatic congestion due to cardiac dysfunction, especially the right heart chambers. Clinically it manifests as shortness of breath, abdominal distension, hepatomegaly, splenomegaly, paroxysmal nocturnal dyspnea, orthopnea and pedal edema. The basic pathology lying behind is the right sided cardiac dysfunction that produces an increase in the preload and back pressure to the hepatic system producing hepatic congestion and ultimately this prolonged hepatic congestion eventually leads to liver cirrhosis. The treatment for the same is based on the management of the underlying cardiac condition causing a hemodynamic imbalance. Herein, we set forth a case report of a 46-year-old lady with abdominal distension, refractory ascites, and recurrent episodes of shortness of breath having prior history of cardiac failure and tricuspid regurgitation.

Corresponding author:** ***Sumol Ratna

Citation: Sumol Ratna, Simran Gangwani, Divyansh Singh, Neha Gurjar, Arshita Gupta, Pragya Dalel, Nishant Sharma, 2024. "How your heart can destroy your healthy liver featuring Cardiac Cirrhosis: A case presentation.*". International Journal of Current Research*, 16, (07), 29402-29404.

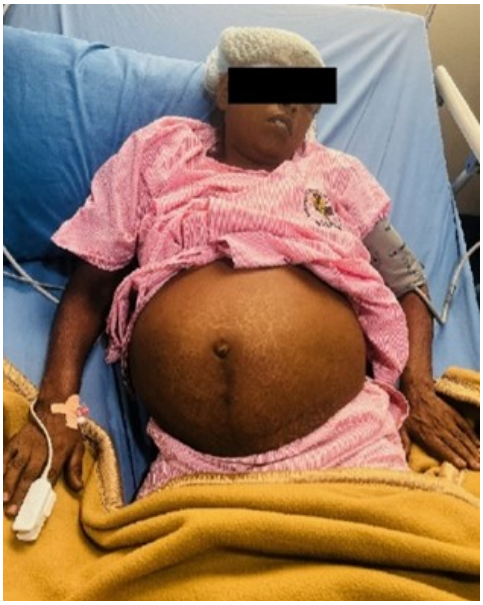
INTRODUCTION

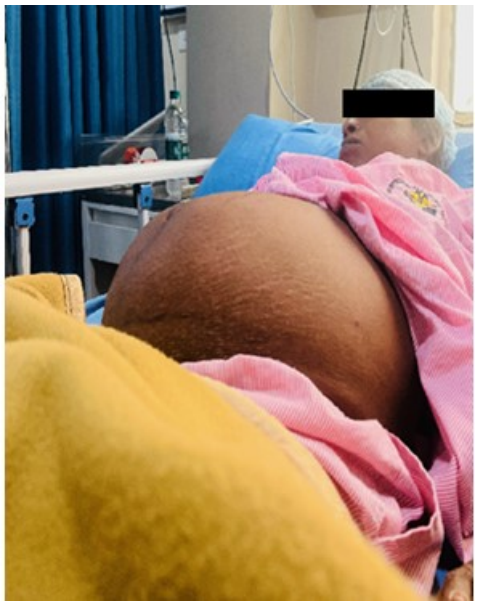
Cardiac cirrhosis is the consequence of the hemodynamic rearrangements caused due to the cardiac failure along with the hepatic dysfunction. Usually, it is the right sided heart pathology that generates right heart failure causing an increase in venous congestion along with increased pressure in the hepatic sinusoids. Valvular abnormalities (tricuspid regurgitation, mitral regurgitation), severe pulmonary hypertension, cor pulmonale, cardiac tamponade, constrictive pericarditis and biventricular heart failure are some of the common cardiac causes precipitating cirrhosis. The pathophysiology behind hepatic dysfunction is either an increase in cardiac filling pressures or low cardiac output and impaired perfusion. An increase in the preload or central venous pressure due to right ventricular dysfunction deteriorates the liver. The elevated pressure is transmitted from the right heart chambers to the hepatic veins and sinusoids, leading to intrahepatic edema, decreased perfusion and oxygen diffusion, as well as hemorrhagic injury and modification of hepatocyte architecture along with collagen deposition and fibrosis of the hepatic veins and sinusoids.

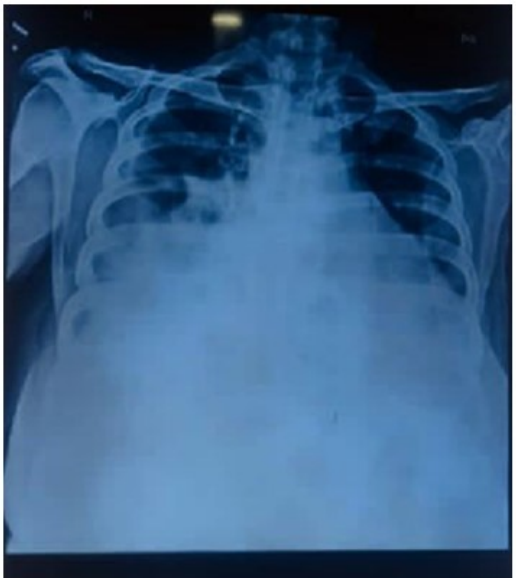
Grossly, a characteristic nutmeg appearance of liver is seen in cardiac cirrhosis. The treatment modalities range from medical treatment to surgical procedures calling intervention of transplantation of either liver, heart, or both. Therefore, in such a scenario, it becomes a huge challenge to diagnose a case of cirrhosis associated with any cardiac abnormality.

CASE REPORT

A 46-year-old lady presented to the emergency with a month history of abdominal distension along with dyspnea since past 20 days. She had no complains of abdominal pain/lump, vomiting, diarrhea, decreased urine output, hematuria, facial puffiness, fever or blood transfusion. She was diagnosed with dilated cardiomyopathy and tricuspid regurgitation last year. She is not a known case of hypertension, tuberculosis, asthma but is a known case of diabetes and hypothyroidism for which she has been taking medications. She was neither a smoker nor an alcoholic. She experienced lack of sleep and appetite with normal bowel and bladder habits. Alsothere is no history of post-menopausal bleeding.







On general examination the patient was cooperative and well oriented with poor nutrition. On physical examination the patient was thin built with pallor, icterus, lymphadenopathy, bilateral pedal edema, engorged neck veins and abdominal distension with everted umbilicus.

Table 1

Lab reports	Results
Hb	13.2gm/dL
TLC	5630/Cumm
Platelet count	227000/mm^3
RBC count	4.33million/mm^3
HCT	39.8%
MCV	91.9fL
MCH	30.4pg
MCHC	33.1g/dL
RDW-CV	16.4H %

Table 2

Medications
INJ LASIX IV
TAB ALDACTONE
TAB CARCA 3.125
TAB THYRONORM
TAB ECOSPIRIN AV 75
TAB EPTUS 25
TAB CILACAR
TAB CARDACE 1.25

No Kayser-Fleischer ring was observed on slit lamp examination. Her vitals were stable. She had tense ascites as fluid thrill test comes out to be positive coupled with shifting dullness since note changes from resonant at the midline to dull while moving to the flanks on per abdomen examination. On cardiovascular examination she had a raised JVP and apical impulse displaced 2cm laterally. On auscultation 1st and 2nd hearts sounds were audible with loud pulmonary component of 2nd heart sound. The holosystolic, high pitched blowing murmur of tricuspid insufficiency best heard at lower left sternal border. The holosystolic murmur intensifies with inspiration and decreases with expiration. On respiratory system examination, there were diminished breath sounds in bilateral lower zones. On CNS examination – No focal neurologic deficits or involuntary movements were observed.

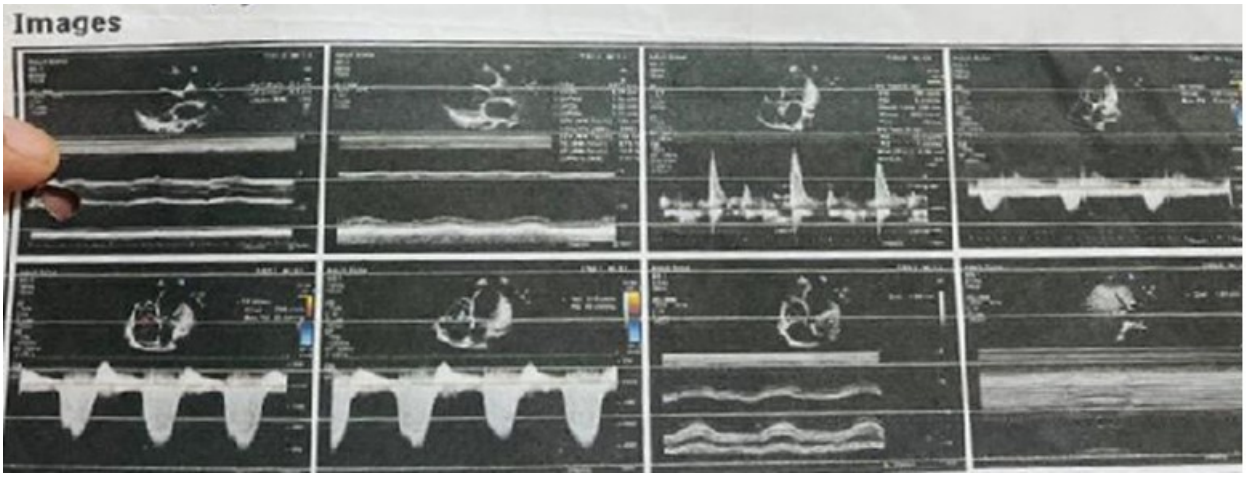
Her hemogram revealed the following findings:

Ultrasound abdomen revealed mild hepatomegaly with coarse echotexture and nodular surface. Portal vein, spleen and gall bladder were found to be normal. Ascitic fluid examination revealed serum ascites albumin gradient(SAAG)– 2.1 g/dL, suggestive of exudative fluid with no pus cells, malignant cells or bacteria. Culture was sterile and Adenosine deaminase (ADA) was normal. Echocardiography revealed severe left ventricular systolic dysfunction with EF = 15-20% along with dilated LA/LV and RA/RV. Mild MR and severe TR was also reported. However, there were no features of pulmonary HTN or pericardial effusion.

DISCUSSION

Liver cirrhosis can be influenced by any right sided pathology of body that may lead to right sided cardiac failure which can result in significant increased venous congestion and increased pressure of hepatic sinusoids as seen in a condition known as cardiac cirrhosis(Congestive hepatopathy). Cardiac cirrhosis is a serious life-threatening disease with high morbidity and mortality rates. The prevalence rate of this disease is not well recorded since this disease can be asymptomatic. The diagnosis may be delayed due to other injuries in theliver.

Treatment was given as follows:



Usually, the mortality rate is depended on underlying cardiac pathology rather than hepatic due to hemodynamic dysfunction. The age presentation for this disease is also obscure since it is completely dependent upon the onset of decompensated heart failure,but as the age progresses, the risk for cardiac induced cirrhosis also increases. The treatment of this disease solemnly depends upon the early diagnosis. Early diagnosis and early intervention can help in the prevention of further progression of the disease. Theinterpretation for these patients is guarded. If the primary underlying causes is not managed well, or there is a delay in diagnosis, the prognosis of these patients is grave. Patients who are to survive this condition end up with multiorgan damage [4][5][6][7].

CONCLUSION

Cardiac cirrhosis is an uneventful condition if not diagnosed at earliest as it can cause significant morbidity and mortality following multi-end organ damage if the patient were to survive. It is best characterized by hyperkinetic circulation causing increased cardiac output and venous congestion. Most of the times, it develops as subclinical condition which makes it difficult to catch the disease. The underlying condition also masks the disease due to multiple liver injuries causing delayed treatment and poor prognosis. Timely diagnosis andearly intervention can help these patients to survive with a good prognosis and have a better way of carrying out their lifestyle [8][9].

REFERENCES

1. Yuan W, Lu HZ, Mei X, Zhang YY, Zhang ZG, Zou Y, Wang JF, Qian ZP, Guo HY. 2019. Cardiac health in patients with hepatitis B virus-related cirrhosis. Medicine (Baltimore). Mar;98(13):e14961.
2. Koehne de Gonzalez AK, Lefkowitch JH. 2017. Heart Disease and the Liver: Pathologic Evaluation. *Gastroenterol Clin North Am.,* Jun;46(2):421-435.
3. M. VanWagner L.B. Lin G. et al. 2020. Redefining cirrhotic cardiomyopathy for the modern era. Hepatology. 71: 334-345
4. Houghton D, Zalewski P, Hallsworth K, Cassidy S, Thoma C, Avery L, Slomko J, Hardy T, Burt AD, Tiniakos D, Hollingsworth KG, Taylor R, Day CP, Masson S, McPherson S,Anstee QM, Newton JL, Trenell MI. The degree of hepatic steatosis associates with impaired cardiac and autonomic function. J Hepatol. 2019 Jun;70(6):1203-1213.
5. Yuan W, Lu HZ, Mei X, Zhang YY, Zhang ZG, Zou Y, Wang JF, Qian ZP, Guo HY. 2019. Cardiac health in patients with hepatitis B virus-related cirrhosis. Medicine (Baltimore). Mar;98(13):e14961.
6. Hussein, M. S., Almujil, S. J., Banheem, A. S. S., Alsuhaymi, N. N., Alhelali, M. H., Aldukhi, D. H. A., Alsaedi, K. R. B., Al-Wagdani, N. M., Almadi, E. A. S., Aladhadh, K. M. A. and AlAjmi, F. A. M. R. H. 2021. "Overview on Cardiac Cirrhosis and Congestive Hepatopathy - A Review", *Journal of Pharmaceutical Research International,* 33(47A), pp. 212–220. doi: 10.9734/jpri/2021/v33i47A33006.
7. S Møller, J H Henriksen, 2009. Cardiovascular complications of cirrhosis, Postgraduate Medical Journal, Volume 85, Issue 999, January,
8. Liu H, Gaskari SA, Lee SS. 2006. Cardiac and vascular changes in cirrhosis: pathogenic mechanisms. World J Gastroenterol. Feb 14;12(6):837-42. doi: 10.3748/wjg.v12.i6.837.

****