A rare cardiac cirrhosis resulting from hypertensive heart failure associated with hepatorenal syndrome and refractory massive ascites in a patient in Orlu, Nigeria

Anyabolu EN1*, EnwereOO1, Agina US1, Anyabolu AE2

1Department of Internal Medicine, Imo State University Teaching Hospital, Orlu, Nigeria. 2Department of Medicine, Nnamdi Azikiwe University Teaching Hospital, Nnewi, Nigeria.

*Corresponding author: enhealer@yahoo.com

Received: 17.11.15; Accepted: 18.12.15; Published: 19.12.15

ABSTRACT

Background: Cirrhosis does not commonly result from cardiac disease. Cardiac cirrhosis associated with hepatorenal syndrome and refractory massive ascites is rare. Intractable massive ascites in this setting usually responds poorly to medical therapy. Aim: This case report documents a rare cardiac cirrhosis associated with hepatorenal syndrome and intractable massive ascites that improved with therapeutic peritoneal paracentesis and albumin infusion in a patient in Orlu, Nigeria. Findings: The patient was a 55 year-old man that was brought as an emergency case with history of fainting attack and loss of consciousness for 5 hours. His random blood sugar revealed he had severe hypoglycemia. He was given bolus 50% Dextrose and further 10% Dextrose, with resolution of the hypoglycemia. Further review and investigations showed he had chronic biventricular heart failure, cardiac cirrhosis, hepatorenal syndrome and refractory massive ascites. Following large-volume peritoneal paracentesis, albumin infusions, diuretics and digitalis therapy, ascites remitted. In three months on admission and two months of follow-up, renal filtration function improved remarkably without vaptans therapy and dialysis. Conclusion: This case report of a rare cardiac cirrhosis resulting from chronic biventricular heart failure, and complicated by hepatorenal syndrome, highlights the need for consideration of therapeutic peritoneal paracentesis with adjuvant albumin infusion and diuretics in the management of the ensuing refractory massive ascites, especially in areas where facilities for shunts are not available and also given the disappointing outcomes of such shunts and high cost of vaptans.

Key words: Albumin, cardiac cirrhosis, hepatorenal syndrome, ascites, paracentesis, dialysis

INTRODUCTION

The world prevalence of cardiac cirrhosis is not known. In Nigeria, its prevalence is also obscure. Cardiac cirrhosis, also known as congestive hepatopathy, is a rare complication of heart failure. Cirrhosis, irrespective of aetiology, can lead to complications that include hepatorenal syndrome and ascites, among others. As ascites resulting from
Cardiac cirrhosis may be massive and distressing. It may have poor response to medical therapy. Outcomes of surgical interventions are disappointing. Furthermore, timely recognition and treatment of a treatable cardiac cause of cirrhosis will lead to resolution of the cirrhosis.

Cardiac cirrhosis associated with hepatorenal syndrome and intractable massive ascites is rare. From literature search, there was paucity of reports on hepatorenal syndrome and refractory massive ascites arising from cirrhosis of cardiac origin in Nigeria.

We thus illustrate a rare cardiac cirrhosis that resulted from biventricular heart failure, and complicated by hepatorenal syndrome and massive ascites that showed remarkable renal filtration function improvement following large volume therapeutic peritoneal paracentesis without the use of vaptans or dialysis.

CASE PRESENTATION

Patient was a 55 year-old man first seen in the Emergency Unit of our hospital, Imo State University Teaching Hospital, Orlu, Nigeria, on account of fainting attacks and loss of consciousness of 5 hours. He had profuse sweating but was afebrile. Pulse rate was 106/min and blood pressure 110/70 mmHg. His random blood sugar, 2.1mmol/l, revealed he has severe hypoglycemia. He was given bolus 30mls of 50% Dextrose and further 1L of 10% Dextrose 8 hourly, with resolution of the hypoglycemia. He regained full consciousness about 60 minutes of commencement of treatment.

Further review in the ward showed he had swelling of both legs of 3 years, abdominal swelling and yellowness of the eyes of 1 year duration. The leg swelling which started about 3 years ago was first noticed on the feet and gradually progressed to the thigh, scrotum and sacrum. Initially, it worsened as the day progressed, and was associated with breathlessness on mild exertion, orthopnea, paroxysmal nocturnal dyspnea and progressive tiredness.

About a year ago, he developed progressive abdominal swelling, jaundice and distressing generalized itching. There was no associated fever. He was a known hypertensive patient diagnosed 12 years ago but was not compliant with the use of anti-hypertensive medications. He was not a known diabetes mellitus patient. He has no risk factors for chronic liver disease, and had no previous jaundice or blood transfusion. Progressive tiredness which started about 3 years ago was so severe that patient has not been able to stand or walk since about a year.

For the above complaints, he was managed in peripheral hospitals. Treatments given to him there included 10 sessions of haemodialysis and transfusion of 4 units of blood. He had also been treated for heart failure. He had been managed for several episodes of hypoglycemia in the past year.

There was no family history of liver, heart or kidney disease. Patient was married and had 21 children. He was a clergy and founder of an African Traditional Religion. There was no history of alcohol, cigarette or substance use.

Physical examination showed he was middle-aged, in obvious respiratory distress, chronically ill-looking, afebrile, pale, and icteric. He had anasarca, but had no asterixis. There was no peripheral lymph gland enlargement. Pulse rate was 88 per minute, full volume and regular. Blood pressure was 160/90 mmHg in supine position. JVP was raised. Apex beat was at the 7th left intercostal space in the mid axillary line. First and second heart sounds (S1 and S2) were heard. The third heart sound (S3) was heard at the apex and left lower parasternal border. Basal crepitations were heard bilaterally. Abdomen was grossly distended. Ascites was demonstrated by fluid thrill. Liver and spleen were not enlarged. Central nervous system examination showed he was conscious and oriented, with no sign of meningeal irritation, no memory loss, no cranial nerve deficit, and no cerebellar sign. However, there was grade 3 power in each lower limb, and grade 5 in each upper limb. There was no dysgraphia. Skin examination revealed diffuse scratch marks, excoriations, and darkened color.

Impression of congestive heart failure on a background of chronic liver disease and chronic kidney disease was made.

The results of the investigations are shown in table 1. Urinalysis was unremarkable. Serum ascitic albumin gradient (SAAG) 1.5., suggested ascetic fluid was transudative. Adenosine deaminase was not done. α-feto protein was normal. Serum calcium and serum phosphate were within normal range. Uric acid was not done. Fasting serum lipid profile was within normal range. Serum electrolytes, urea and creatinine showed evidence of hyponatremia, mild acidosis and azotemia. Liver
function test showed liver enzymes were within normal range. Repeat renal function tests after four weeks showed serum creatinine 173umol/l, urea 8.0mmol/l, and sodium 134. Serological makers of autoimmune liver disease were unremarkable.

Renal sonography showed that both kidneys were normal in size and echo texture, and maintained good cortico-medullary differentiation. Computerized tomogram showed that the liver appeared normal and its enhancement was normal. There was dilatation of inferior venacava. Gall bladder appeared distended. Common biliary duct appeared prominent with maximum diameter of 13mm with smooth tapering till terminus. Gross ascites was noted. Extensive arterial calcifications were noted. Degenerative changes were seen in the spine. Kidneys were normal in size and both showed normal enhancement. No calculi, hydronephrosis or cysts were seen. Echocardiography revealed that both ventricles showed severe systolic dysfunction; ejection fraction was 36%. There was dilatation of both ventricles. Mitral and tricuspid regurgitation was present. There was no pericardial effusion, and no clots. We did not do cardiac catheterization as the facility was not available. However, liver biopsy was not done because the patient did not give consent.

Diagnosis of cardiac cirrhosis, complicated by hepatorenal syndrome and refractory massive ascites was made.

Treatments given included amlodipine 10mg daily, digoxin 0.125mg daily, spironolactone 75mgbd, frusemide 40mgbd, livolyn capsule I daily, and therapeutic peritoneal paracentesis with adjuvant albumin infusion. For each litre of ascitic fluid removed, 8g of albumin was infused intravenously. Overall, 120g of albumin was used and peritoneum became dry. Two units of blood were transfused. He was placed on low-salt diets. Dietary protein was maintained at 1g/kg/day. Patient did not receive any dialysis or vaptans therapy all through the management. After about three months on admission, he was discharged home, and followed up in the clinic for 2 months. He was lost to follow-up. During the follow-up, he was stable and has complete remission of the ascites and marked appreciation in renal function.

DISCUSSION

Liver disease can result in cardiac disease, and cardiac disease can cause liver disease. However, cardiac cirrhosis is more commonly seen than diseases of the heart attributable to liver aetiology. Hypertension, irrespective of aetiology, can cause biventricular failure which may be associated with volume overload and low cardiac output. These may result in congestive hepatopathy, usually from right heart failure, and hepatic ischemia, usually from left heart failure, with potential to cause hepatic fibrosis and subsequently, hepatic injury. Cirrhosis may develop from hepatic fibrosis. Portal hypertension may be associated with ascites, hepatorenal syndrome, among others.

Our patient has congestive cardiac failure and congestive hepatopathy. The history of hypertension of 12 years, clinical features and imaging evidence of chronic heart failure of 3 years, jaundice, generalized pruritus, dominant massive ascites more than leg edema of the egg and stick appearance, and computerized tomogram evidence of inferior venacava dilatation, all supported the diagnosis of cardiac cirrhosis in this patient.

Liver biopsy was not done to establish tissue diagnosis of cirrhosis. However, the histology of the liver in congestive hepatopathy may be normal, consigning diagnostic instruments to clinical evidence of chronic liver disease, heart failure and imaging evidence of inferior venacava and hepatic vascular dilatation, suggesting hepatic congestion.

Mild elevation in serum aminotransferases, AST/ALT ratio greater than 2.0, and serum total protein and albumin within normal range were seen in our patient. Cardiac cirrhosis may be associated with normal or mildly elevated liver enzymes as was the case in our patient. However, liver enzymes were found to be high in one case report. The high AST/ALT ratio in our patient might suggest cirrhosis of alcohol aetiology. However, he has no history of alcohol consumption. Prothrombin time was normal in our patient. A report has also shown a similar normal prothrombin time in cardiac cirrhosis.

Hepatorenal syndrome, which may present as acute kidney injury, or acute kidney injury in the setting of chronic kidney disease, may complicate cirrhosis. We observed that this patient received about 10 sessions of hemodialysis over 2 years prior to presenting to us. Following management, his serum creatinine decreased to 173mmol/l, down from 483mmol/l; he also has low urine sodium. This and sonogram evidence of normal renal size and structure suggested the patient has type 2 hepatorenal syndrome. Although chronic kidney disease in this patient could also be adduced to...
longstanding hypertension, the marked improvement in renal function following peritoneal paracentesis is suggested azotaemia of hepatorenal syndrome.

Table 1: Results of laboratory investigations

<table>
<thead>
<tr>
<th>Investigations</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full blood count</td>
<td>Hb: 7.6g/dl, Platelets: 186x10⁹ cells/ml, WBC: 7400 cells/ml, Neutrophils: 54%, Lymphocytes: 44%, Eosinophils: 0%.</td>
</tr>
<tr>
<td>Random blood sugar</td>
<td>5.2mmol/l.</td>
</tr>
<tr>
<td>Serum Protein</td>
<td>Total: 7.2g/dl, Albumin: 3.9g/dl</td>
</tr>
<tr>
<td>Ascitic fluid</td>
<td>Sugar: 4.7mmol/l, albumin: 2.6g/dl, Serum ascitic albumin gradient (SAAG): 1.5. Ascitic fluid showed no pus cells, no bacteria, no malignant cells, and no bacterial growth on culture</td>
</tr>
<tr>
<td>α-fetoprotein</td>
<td>6.0iu (&lt;10 iu).</td>
</tr>
<tr>
<td>Serum calcium</td>
<td>1.2mmol/l</td>
</tr>
<tr>
<td>Serum phosphate</td>
<td>1.14mmol/l</td>
</tr>
<tr>
<td>Fasting serum lipid profile</td>
<td>Total cholesterol: 2.3mmol/l, low density lipoprotein cholesterol: 1.2mmol/l, high density lipoprotein cholesterol: 0.8mmol/l, Triglyceride: 0.7mmol/l,</td>
</tr>
<tr>
<td>Liver function tests</td>
<td>Total bilirubin: 22iu/l, conjugated bilirubin: 4iu/l, aspartate transaminase: 89 iu/l, alanine transaminase: 28 iu/l</td>
</tr>
<tr>
<td>Viral screening</td>
<td>HBsAg: negative, HCV: negative, HIV I and II: negative</td>
</tr>
</tbody>
</table>

The management of intractable massive ascites in cardiac cirrhosis includes initial diuretic therapy, usually a combination of aldosterone antagonist like spironolactone and loop diuretic like frusemide. Ascites not yielding to medical therapy would require large volume paracentesis, and salt-poor albumin infusion. Our index patient had very good response following peritoneal paracentesis. In exchange, infusion of 8g of albumin was given for each litre of ascitic fluid removed, in addition to diuretic therapy. After sessions of paracentesis to dryness, patient was stable for over 12 weeks, during which period ascites did not recur. He was lost to follow-up and so we could not assert the duration the relief lasted. We did not also use
vaptans which are indicated in hepatorenal syndrome associated with massive ascites resulting from cirrhosis.^[1] This contrasted with case reports in which vaptans and paracentesis were used before ascites could resolve.[^1]

Cardiac cirrhosis traceable to heart failure of a treatable cause usually resolves when the cause of the heart failure is timely treated.[^4][^5] Chandra et al. reported a case of cardiac cirrhosis resulting from heart failure due to a congenital heart disease in which they observed that the cirrhosis resolved following heart repair surgery for the defect.[^8] Expectedly, the treatment target for our patient was alleviation or complete relief of symptoms of heart failure and congestive hepatopathy, as congestive cardiac failure due to hypertension cannot be cured.

**CONCLUSION**

This case report showed a rare case of cardiac cirrhosis associated with hepatorenal syndrome and refractory massive ascites in a patient in Orlu, Nigeria. It highlights the need for re-consideration of large volume paracentesis for refractory massive ascites with albumin infusion in patients with cardiac cirrhosis, hepatorenal syndrome and refractory massive ascites, given the unavailability and high costs of vaptans and shunt surgery, and the disappointing outcomes of shunt surgery.

**REFERENCES**


Submit your valuable manuscripts to Michael Joanna Publications for:

- User-friendly online submission
- Rigorous, constructive and unbiased peer-review
- No space constraints or colour figure charges
- Immediate publication on acceptance
- Unlimited readership
- Inclusion in AJOL, CAS, DOAJ, and Google Scholar

Submit your manuscript at

www.michaeljoanna.com/journals.php

doi: http://dx.doi.org/10.14194/ijmbr.4.3.6

How to cite this article: Anyabolu EN, EnwereOO, Agina US, Anyabolu AE. A rare cardiac cirrhosis resulting from hypertensive heart failure associated with hepatorenal syndrome and refractory massive ascites in a patient in Orlu, Nigeria. Int J Med Biomed Res 2015;4(3):149-153