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(CASE REPORT)

A case of acute decompensated CCF with massive bilateral pleural effusion that required prolonged chest tube drainage in a tertiary facility in Nigeria

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Abstract

Heart failure with massive bilateral pleural effusion (over 2 liters) is a rare occurrence that is yet to be dispelled due to the dearth of information on the topic. The prevalence and mortality of CCF are rising. Clinical features of heart failure include dyspnea, orthopnea, fatigue, anorexia early satiety, pedal edema, abdominal distension/ascites, anorexia, nausea, elevated jugular venous pressure, displaced apex beat, crepitations, hepatomegaly, jaundice. Pleural effusion may complicate this picture and worsen the clinical state. Pleural effusion in heart failure is usually transudative, with pleural fluid parameters in keeping with the Light criteria. The case is that of a 59-year-old man with acute decompensated congestive cardiac failure 2^0 to Dilated cardiomyopathy in NYHA class IV precipitated by poor drug adherence, complicated by massive bilateral pleural effusion. He required prolonged chest tube drainage and subsequently had good clinical improvement. This case report serves as a wake-up call that these essentially rare occurrences still exist and that the clinical judgment and expertise of the clinician will be tested on a daily basis.

Keywords: Acute Decompensated CCF; Massive Bilateral Pleural Effusion; Prolonged Chesttube Drainage; Dilated Cardiomyopathy; Tertiary Facility in Nigeria.

1. Introduction

Heart failure is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood[1]. Pleural effusion(PE), the pathological accumulation of fluid in the pleural space has been linked to congestive heart failure, which in itself is recognized as a cause of transudative effusion accounting for 37.4% of cases in the USA[2,3]. Also, congestive heart failure is the most common cause of bilateral pleural effusion with values ranging between 51-61%[2,3,4,5]. Mortality rate is still on the increase with CCF± pleural effusion with a one-year mortality of 57% and a 30day mortality rate of 19.2 [3,6]. Massive pleural effusion is defined as fluid collection on chest radiograph that appears as complete or near-complete opacification of the ipsilateral thorax.[16] Massive pleural fluid collection is commonly caused by malignancy.[16] Rarely does heart failure cause massive bilateral pleural effusion requiring drainage as most pleural effusions resolve spontaneously with anti-heart failure management[1,7]. Timely thoracentesis or the insertion of a pleural drain is necessary if a pleural effusion is large[8].

2. Case presentation

Index case is a 51 yr old male civil servant who presented with complains of recurrent bilateral leg swelling of 2 years duration and recurrent difficulty in breathing of 2 years duration with index episodes of presenting symptoms starting 2weeks prior to presentation. Associated history of abdominal swelling, orthopnea, paroxysmal nocturnal dyspnea,

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effort intolerance and nocturnal cough. He is a known hypertensive of 5yrs duration with poor drug adherence however, not a diabetic. He is married in a monogamous setting with 3 children.

Examination findings was that of a middle aged man, acutely ill-looking, in respiratory distress, afebrile,not pale, anicteric, acyanosed, not dehydrated and bilateral pitting pedal edema.
pulse rate was 108bpm irregularly irregular with thickened arterial wall and locomotor brachialis and distended neck veins. Blood pressure was 120/80mmhg, Diffused,non heaving apex beat.
Heart sound was S1 and S2 with pansystolic murmur loudest at the apex.

Respiratory rate was 30c/m, SpO2 -90% in room air.
Tracheal was central with equal chest expansion bilaterally. Percussion note was stony dull in middle and lower lung zone bilaterally with reduced breathe sound and vocal resonance in middle and lower lung zone bilaterally.

Abdomen was distended, moved with respiration with mild right hypochondrial tenderness. Liver was 6cm below right coastal margin with a span of 16cm, soft and smooth. S^0K^0 with ascites demonstrable by shifting dullness.
He was conscious and alert, OTPP pupils were equal, normal size and reactive to light bilaterally. There was no obvious CN deficit. Muscle bulk, tone and Reflexes was normal globally. Power was 5/5 globally.

Ordered investigations came out below:

- E/U/C(mmol/L)
- Na- 133
- K- 4.1
- CL- 104
- HC03 -17
- Urea- 61.2
- Cr- 1.54.
- FBC
- HB- 13.2
- pcv- 40%
- Twbc- 4770
- N- 51.9
- L- 31.4
- plt- 190,000
- Urinalysis
- Appearance - straw color
- SG- 1.015
- RBC- 1-5
- pus cells- 1-5
- Blood- Trace
- pH- 6.0
- others- normal
- LFT
- Albumin- 35
- ALP- 135
- ALT- 14
- AST- 37
- Total Bil- 57.81
- Conj. Bil-37.36
- Unconj Bil-
- Total protein- 73
- GGT- 132
- Globulin- 38
- Alb/Glo- 0.92
- Echo scan- revealed very poor Left ventricular contraction with ejection fraction of 19%
- ECG- Sinus tachycardia and Left ventricular hypertrophy.

2.1. Abdopelvic scan

- Revealed findings of hepatomegaly with hepatic congestion with ? background chronic liver disease.
- Bilateral acute Renal parenchymal disease(grade2)

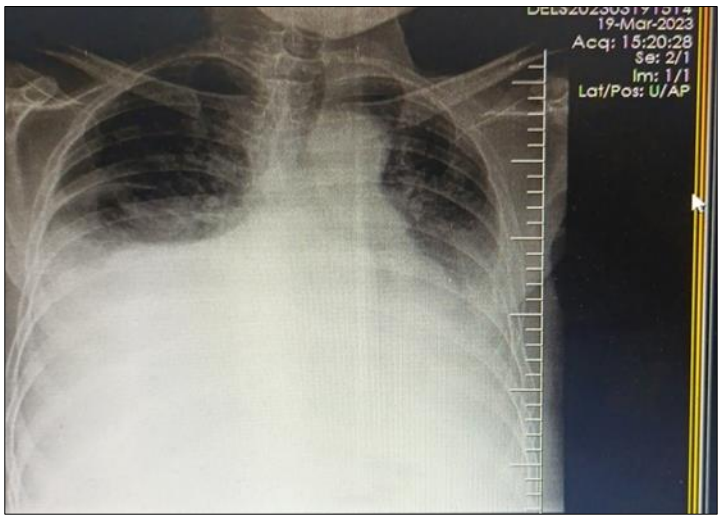

Figure 1 Chest Radiograph showing bilateral massive pleural effusion at presentation

CXRAY- revealed homogeneous opacity with lateral meniscus involving the right and left mid and lower hemithorax. Heart size could not be objectively assessed with conclusion of bilateral pleural effusion.

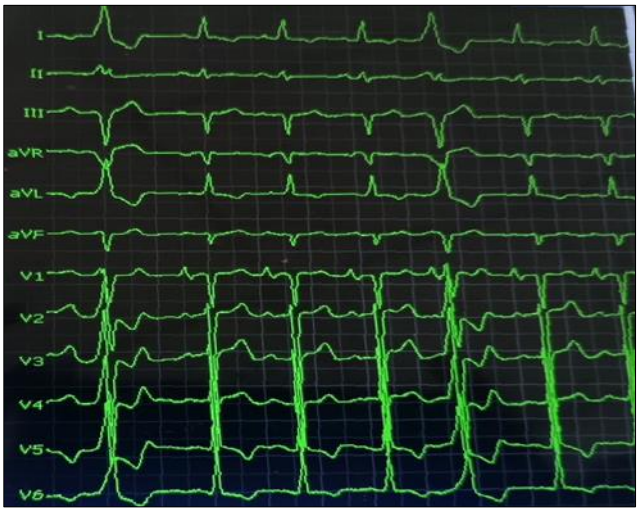

Figure 2 Electrocardiogram of patient at presentation

A assessment of Acute Decompensated CCF 2^0 to DCM in NYHA IV ppt by poor drug adherence complicated by massive bilateral pleural effusion was made.

Also, emergency care and medications were commenced in relation to the GDMT guideline; Intravenous frusemide,Tabs Lisinopril, Tabs spironolactone, Tabs digoxin, Tabs vitC, Caps MIM Subcutaneous Enoxaparin. Other measures instituted included; oxygen by nasal prongs and to ensure SpO2 ≥ 93%, counselling patient and relatives on diagnosis and need for treatment compliance, dietary counseling, daily weighing, strict monitoring of input and output chart, for him to be nursed 45^0 headup.

Subsequently, Tabs metolazone was commenced which he continued for somedays with no clinical improvement and subsequently had bilateral pleural space tap, aspirating 1litre from the (R) and 250mls of serous fluid from the (L) pleural spaces respectively.

Repeat CXRAY and unimproving clinical state necessitated the decision for chest tube drainage which he subsequently had in the Left hemithorax while on the antifailure regimen with further drainage of about 1L of serosanguinous fluid. Pleural fluid investigations revealed

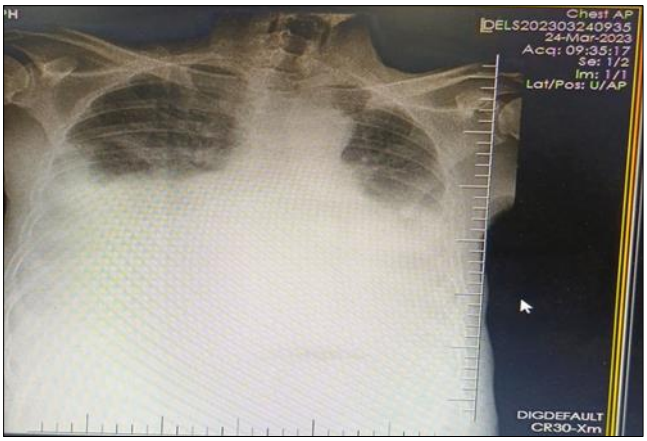

Figure 3 Chest Radiograph showing bilateral massive pleural effusion after use of antifailure regimen

- Pleural fluid protein - 20.5 g/l Transudate range)
- Pleural fluid microscopy
- Leucocytes - numerous
- Organisms - none
- Lymphocytes - 480 /mm^3
- Neutrophils - 160 /mm^3
- Gene xpert
- MTB not detected
- Adenosine Deaminase- 6 u/l (N)
- Lactate dehydrogenase - 244 u/l (E)

He spent a total of 32DOA and showed marked clinical and radiologic improvement with x-ray before discharge.

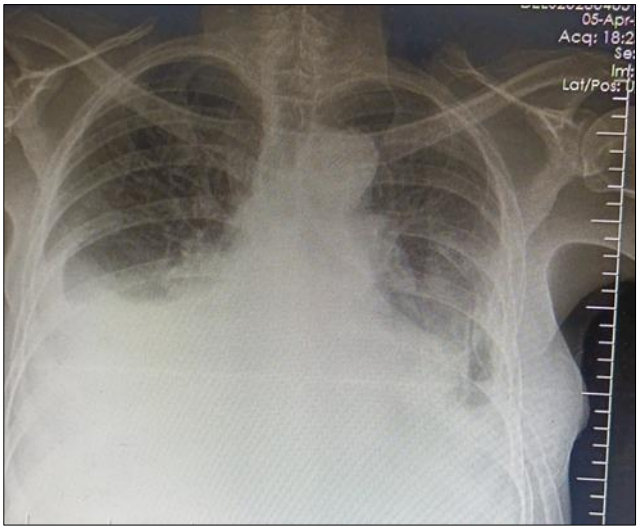

Figure 4 Chest Radiograph showing resolved pleural effusion before patient was discharged

Since discharge, he has had one clinic follow-up. He has remained in his Chronic Stable Heart failure state which is evident by repeat Echo scan. Repeat CXRAY still reflected clinical improvement but however still have some residual fluid in his L lower kung zone.

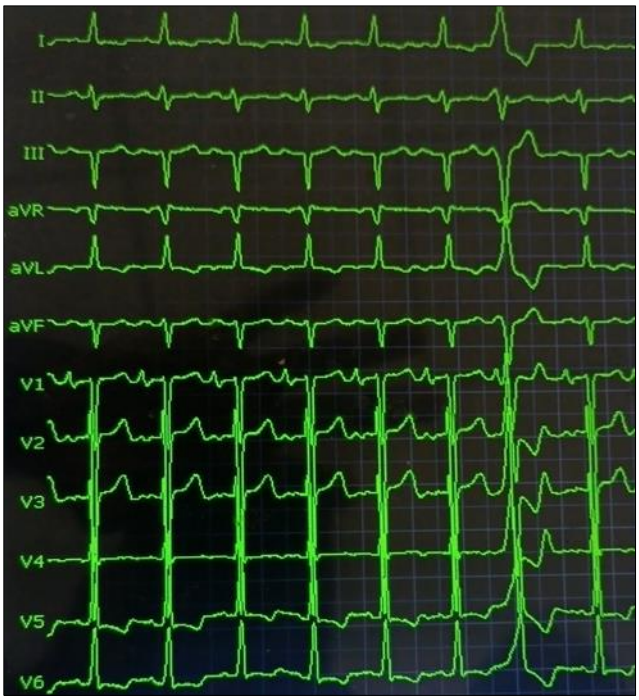

Figure 5 Electrocardiogram of patient at first clinic follow-up

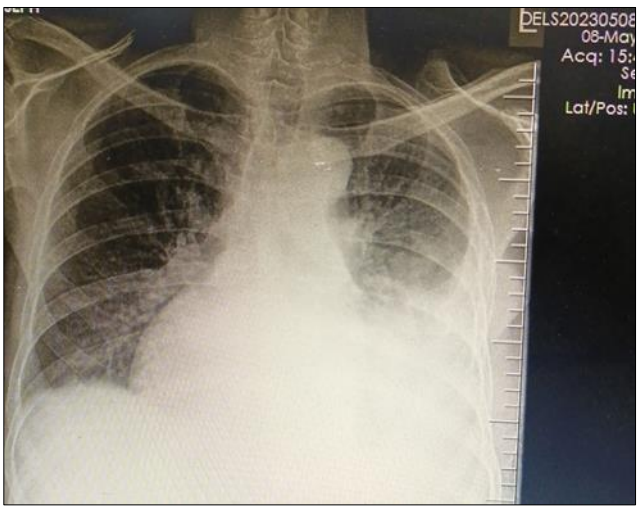

Figure 6 Chest Radiograph of patient at first clinic follow-up

3. Discussion

Heart failure is a chronic dynamic disease and based on this, patients can be logically classified as Chronic Stable Heart failure and Acute decompensated heart failure. While the chronic stable heart failure refers to their stable state even though they are in failure, the acute decompensation refers to a sudden dip from this stable state usually following a precipitant such as febrile illness, poor drug compliance, anaemia and a host of other factors.

3.1. Pathophysiology of heart failure

Heart failure is a hemodynamic disorder and the pathophysiologic basis is the interplay between cardiac output, cardiac contractility, filling pressures, wall stress during systolic and diastolic function and neurohumoral activation[9,10].

Due to the anatomical and functional relationships, PE in patients with CHF is mainly related to left ventricular failure. Also, the number of patients with left ventricular dysfunction or dysfunction of both ventricles is significantly higher than the number of patients with right ventricular failure[11]. Therefore, in daily clinical practice, the vast majority of PEs in patients with CHF is related to left ventricular dysfunction[5].

3.2. Pathophysiology of pleural effusion in heart failure

These effusions usually develop in both the right and left hemithorax but can be unilateral. The pathogenesis involves increased fluid transfer from parietal pleural capillaries into the pleural space and possibly decreased pleural fluid uptake into parietal pleural lymphatic structures. The increased fluid transfer develops due to increased capillary pressure secondary to elevated venous outflow pressure and secondary to decreased lymphatic flow into central vessels secondary to heart failure[12].

3.3. Clinical features of CCF

Symptoms include

- Shortness of breath/dyspnea
- Orthopnea
- PND
- Fatigue, weakness, lethargy
- Leg swelling
- Abdominal distension
- Anorexia, nausea, early satiety.

Signs encountered are

- Cachexia
- Chynes stokes breathing pattern
- Pulsus alternans
- Elevated JVP
- Displaced AB
- Crepitations
- Hepatomegaly, jaundice
- Ascites
- Edema.

Both signs and symptoms are summarized by the Framingham criteria which is a diagnostic tool used in clinical practice[13]. The FC-HF consists of major (paroxysmal nocturnal dyspnoea, orthopnea, jugular venous distension, third heart sound, cardiothoracic ratio >0.5 on X-ray, pulmonary oedema on X-ray, and pulmonary crackling rales) and minor [peripheral oedema, nocturnal cough, dyspnoea at exercise, hepatomegaly, pleural effusion, and tachycardia (defined as ≥100 bpm)] criteria. To fulfil the FC-HF, the presence of two major or one major + two minor criteria is required[13]. The index case met this criteria.

Some patients complain of a dry cough, which can be explained as a manifestation of pleural inflammation or lung compression due to a large effusion[14]. Pleural effusions can also markedly impair the quality of sleep[14].

Pleural effusion has been found to be more common in males, and our case study is male[4].

While the CBC was otherwise within reference range, the metabolic panel was mildly deranged. This can partly be explained by the fact that he was immobile and also dehydrated and reduced renal perfusion. On the other hand, the LFT was in keeping with hepatic congestion which can be inferred originated from the heart failure.

Our case study was in keeping with a transudate which also was observed by Ekpe EE et all in their study that showed transudates in 90% of the cases, while exudates was discovered in only 10% of the cases[7]. However, it is possible the elevated LDH we got from the result are from traumatized red blood cells from the procedure of the chest tube placement which would've been eliminated if serum albumin to pleural fluid albumin gradient or measurement of N-terminal pro-brain natriuretic peptide in the pleural fluid were done[15]. The absence of organisms ruled out possible infectious cause of the pleural effusion.

Outstandingly, in a study by Ekpe EE et al, all the five (100%) patients whose pleural effusion was more than 2000mls had significant symptomatic relief upon drainage of the pleural effusion[7] which was seen in our case study. Reason behind this is not fully understood however, a possible scientific explanation is with pleural effusion drainage, there is (1) reduced lung compression and subsequent improved aeration and oxygen delivery to body tissues (2) reduced production of inflammatory cytokines, acute phase reactants and stress hormones (3) Both factors result in rapid healing of injured tissues.

Although this patient showed marked clinical and radiologic improvement on/after discharge from inpatient care, what is not clear is why he has residual fluid in chest but it only can infer the severity and peculiarity of this patient who required a very long time for chest tube drainage after an initial pleural space tap to achieve clinical improvement.

Limitation

Serum albumin to pleural fluid albumin gradient or measurement of N-terminal pro-brain natriuretic peptide in the pleural fluid was not done as it is expensive and not readily available. Other investigations not done include TFT and cardiac catheterization.

4. Conclusion

CCF still remain a major cause of morbidity and mortality the world at large and maybe complicated with massive bilateral pleural effusion. This patient has Ef of about 19% and with further follow-up will benefit from newer innovations in medical practice such as implantable defibrillator, cardiac resynchronization therapy (in event there's widened QRS complex), chronic anticoagulation therapy and at the extreme, a heart transplant which is expensive and readily not available especially in poor resource setting such as this. CCF and it's attendant complications remain an area in cardiology still very open to research and newer innovations and can not be overemphasized.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest.

Statement of ethical approval

Ethical approval was sought and obtained.

Statement of informed consent

Informed consent was obtained from the patient.

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