Tropical Journal of Nephrology Vol.5 No.2 December, 2010 121- 124 *Case Report*

Occurrence of Rheumatic Heart Disease and Acute Glomerulonephrtis in an African Child

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ABSTRACT

Rheumatic heart disease (RHD) and acute glomerulonephritis (AGN) rarely co-exist. Only few occurrences have been reported in the literature with none among blacks of African descent. We report a 13 year old Nigerian girl who presented in our hospital with 3 month history of cough, orthopnoea and breathlessness both on exertion and at rest and three weeks history of bilateral pitting oedema up to the sacrum from the foot. There was previous history of feet swelling about 5 years prior to this presentation which resolved without hospitalization. There was associated raised jugular venous pressure and pan systolic murmur radiating to the axilla with pulmonary accentuation. Blood pressure of 170/ 110mmHg at admission. Proteinuria and haematuria were 3+ and 2+ respectively at admission, however serum protein, albumin and triglyceride were within normal range. Serum creatinine was on the upward trend from 491µmol/l at admission. Erythrocyte sedimentation rate was 45mm/hr. Urine output was between 0.3-1.1mls/kg/hr and the oedema never really subsided. Echocardiography suggested rheumatic heart disease and mitral incompetence. In view of the presence of cardiac symptom and increased erythrocyte sedimentation rate with oedema, hypertension, azotaemia and previous history of body swelling, a suspicion of RHD and AGN is entertained. These remain rare combinations with few reports in the literature and in Blacks living in Sub Saharan Africa.

Keywords: *Rheumatic heart disease; Acute glomerulonephritis; African child*

INTRODUCTION

Acute Rheumatic fever and acute glomerulonephritis rarely if ever occur in the same patient at the same time.[1]However the coincident occurrence of rheumatic fever and glomerulonephritis has been described.[2-4] There are few occasional patient reports of the simultaneous occurrence of the two in the literature with none known to us in black children living in Africa.[5-8]

Case report

We report a 13 year old Nigerian girl who presented in our hospital with cough, breathlessness and

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orthopnoea of 7 months duration. Leg swelling started 2 weeks before presentation. Cough became productive of white frothy sputum with haemoptysis. No history of contact with an adult with chronic cough. No preceding sore throat or skin rash. Breathlessness occurred both on exertion and at rest with easy fatigability. Two weeks to presentation, swelling of both feet occurred with no facial swelling. There was associated intermittent vomiting which contained

and liver span was 13cm. Liver was 6cm below the costal margin. It was firm and tender. She was alert and had no sign of meningeal irritation. She had no cranial nerve deficit, choreform movement, erythema marginatum or polyarthritis.

Echocardiography revealed thickened and clubbed mitral valve but with good motion. The left atrium and ventricle were grossly dilated (Fig.1). Pulse wave spectra display revealed regurgitant jets in the

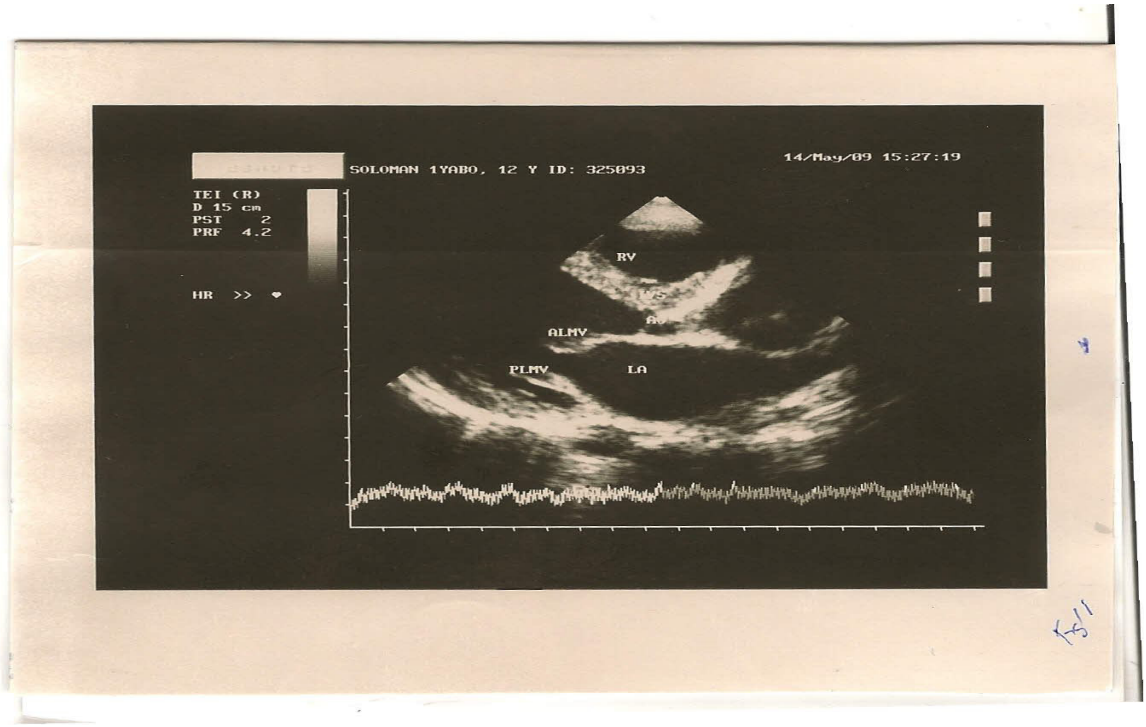

Fig. 1: A two dimensional view of the echocardiographic study in the patient showing dilatation of both left atrium and ventricle and thickened anterior and posterior leaflets of the mitral valve.

recently ingested feed. She has not attained menarche. There is associated dull retrosternal pain that radiated to epigastric area. No history of bee sting or use of mercury containing soap. She is 2nd of 4 children of parents in a monogamous family. Patient had similar illness (swelling of feet 5 years earlier but was not admitted to a hospital).

On examination, she was acute ill looking, mildly distressed, bilateral pitting pedal oedema, up to the sacral region. Pulse rate was 105/min full, regular and good volume. Blood pressure was 170/110 mmHg. Jugular venous pressure (JVP) was elevated to the root of the neck. Grade 3/6 pan-systolic murmur which was maximum at the apical region and radiated to the axilla. Apex beat was at the 5th left intercostals space lateral to the mid-clavicular line with accentuation of P2 with a left parasternal heave. There was tender hepatomegaly

left atrium with a velocity of 4.17m/s translating into a gradient of 69.5mmHg (Fig.2). All these were suggestive of mitral valve disease (mitral incompetence) secondary to rheumatic heart disease (RHD). Chest radiograph showed cardiomegaly. Lentiviral screening and throat culture were negative. Serum creatinine was 491µmol/l and urea was 19.7 mmol/l, protein was 72g/l, albumin was 45g/l, and cholesterol was 4.1 mmol/l ,triglyceride was 1.5 mmol/l potassium was 4.9mmol/l. Urinalysis at admission was protein 3+ and blood 2+. Erythrocyte sedimentation rate was 45/min. Assessment of RHD with mitral incompetence in heart failure and Acute glomerulonephritis (AGN) was made. She was nursed in cardiac position and commenced on parenteral frusemide , oral spironolactone and captopril.

Blood pressure was controlled from 7th to the 22nd day of admission. Oedema also resolved over the same period, however cardiac murmur persisted.

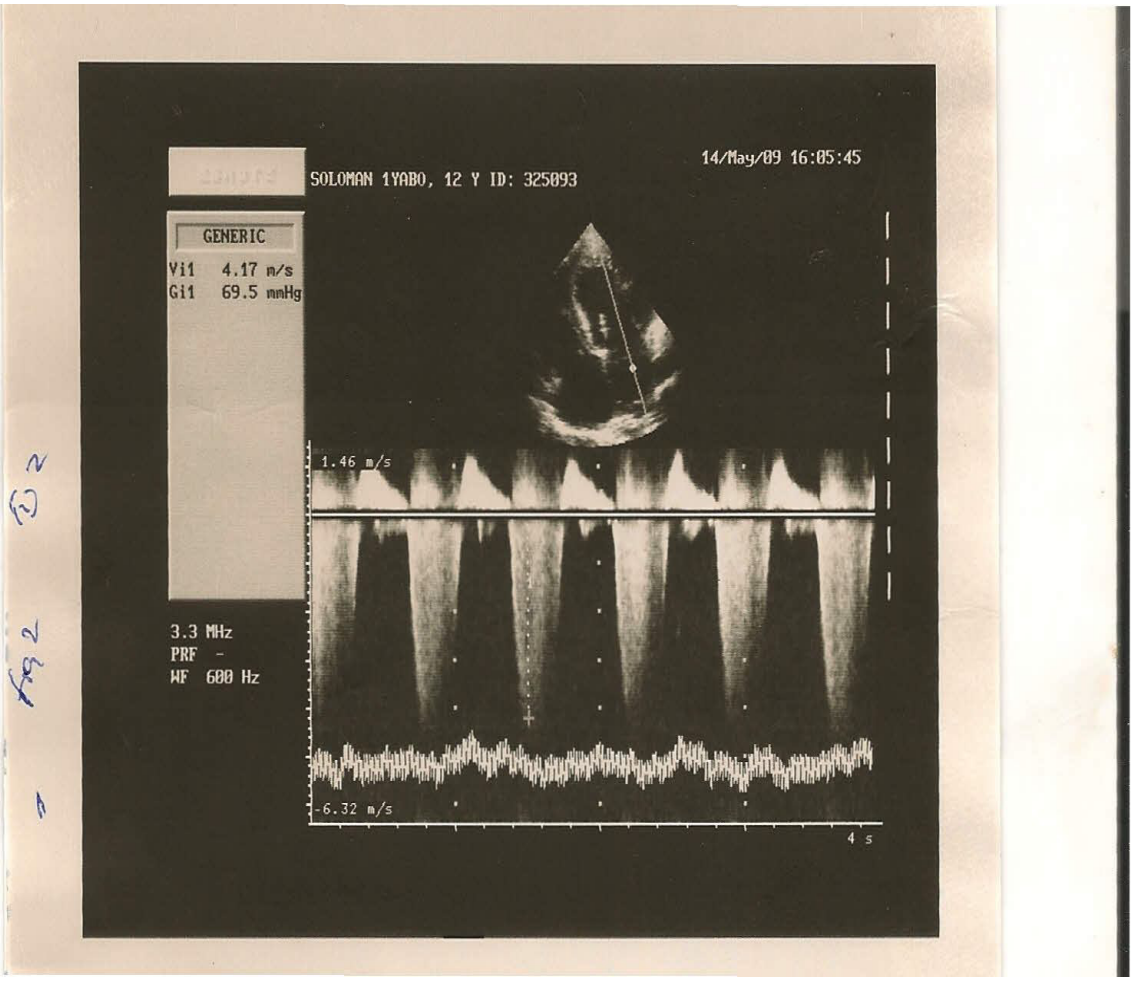

Fig. 2: Shows the spectra display of the regurgitant jet of mitral valve using the continous wave (velocity of 4.17m/s translating into a gradient of 69.5mmHg.)

Urinary output over the period ranged from 0.3-1.0ml/kg /hr. Hypertension and oedema recurred 23rd day on admission and blood pressure ranged between 120/90-150/120mmHg. Serum creatinine was 700µmol/l and dialysis was offered but it was turned down due to financial constraints. Proteinuria reduced to 2+ with no haematuria. As of the time of self discharge on the 29th hospital day, pedal oedema continued to increase with the weight being 42kg when she was last seen. The weight at admission was 39kg, while mean weight throughout the 29 days of hospital stay was 40 kg.

DISCUSSION

The features of AGN and RHD were present in our index patient. For the acute glomerulonephritis (AGN), hypertension, oedema, haematuria and azotaemia were present. The rheumatic heart disease (RHD) was evident by the clinical and echocardiographic evidence of mitral incompetence (Figures 1 and 2).

The occurrence of AGN and RHD are few in the literatures. [5-8] Most of those reported have been among Caucasians and blacks living in the western world and none has been reported in Sub Saharan Africa. Our index patient is the fifth reported case.

The RHD appeared to have antedated the AGN in this patient in view of the presenting history of cough, breathlessness and orthopnoea 7 months prior to the leg swelling. This is in keeping with the findings of other observers who have noticed that when RHD and AGN occurred together, RHD has been the presenting clinical condition followed by the AGN. [5, 7-8]. The reverse occurred in only one case [6]. However, a few setbacks in diagnosis peculiar to the African environment make this case not full proof when compared to similar reported cases. These include the absence of the ASO titre, C3, negative throat culture result (which is not surprising) and lack of renal biopsy.

Not all rheumatic fever patients have a history of preceding upper respiratory tract infections (URTI) and not always are streptococci found on throat culture at the time of acute rheumatic fever, Since the organism often disappear from the pharynx during the 2-5week latent period between the URTI and the onset of rheumatic fever. However, an elevated streptococcal antibody titer can be found in virtually all patients during the acute stage. That notwithstanding, major ingredient that aids the diagnosis of both disorders is present such as mitral incompetence for RHD and oedema, hypertension, haematuria and azotaemia for AGN. It is a known fact that in some patients with established post-streptococcal acute glomerulonephritis (PSAGN), a negative throat culture is not unlikely.[9] Sore throat occurs frequently here especially during the dry season, so it may be difficult remembering a particular episode.

In view of the clear evidence of a RHD in this patient, it might be that the renal involvement are in keeping with possible renal lesions associated with chronic rheumatic heart disease such as focal proliferative glomerulonephritis, mesangial proliferative glomerulonephritis and interstitial nephritis.[4]

Unfortunately, in our patient, renal biopsy was deferred in view of the unstable clinical condition of the patient. Furthermore, she never waited to receive improvement before she was discharged against medical advice. However, the symptom combination of hypertension proteinuria, haematuria and oedema in our patient would presumably have shown PSAGN if a renal biopsy was carried out.

It might be likely that we were dealing with a recurrence of an acute rheumatic fever (ARF) with the latest episode been associated with PSAGN. However, there was no complete evidence of ARF in our patient (Only RHD and elevated ESR were present). Acute rheumatic fever may recur with any M serotype of group A â haemolytic streptococcus. It follows only pharyngitis, but may occur with any M serotype of group A streptococci, while PSAGN is associated with a limited number of M serotype nephritogenic strains type 12 and 49 and may follow both pharyngitis and skin infection. [10-11] Indeed, only about 15 of more than 80 known M types of streptococcus group A are nephritogenic. The pathogenesis is not well understood, but it seems that in a genetically susceptible host, streptococcal tissue injury activates the host's immune system, but because of cross-reactivity and /or molecular mimicry, the immune response results in damage of host tissues. [11]

Our patient illustrates the importance of recognizing that renal disease may occur as part of RHD. The presence of haematuria and proteinuria in a patient with RHD should alert the Physician to monitor the patient more closely regardless of whether the condition is due to RHD alone or to coexistent PSAGN. Furthermore, unlike some other rheumatic scenario that is uncommon in blacks living in Africa, this combination as depicted in our case could occur in blacks living in Sub Saharan Africa.

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