Pulmonary Tuberculosis in a Patient with Tetralogy of Fallot: Timing of Cardiac Surgery – A Case Report

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Abstract: The management of Tetralogy of Fallot with concomitant pulmonary tuberculosis poses significant challenges, especially regarding the optimal timing for cardiac surgery. Currently, no consensus or algorithmic guidelines exist regarding screening for pulmonary tuberculosis in this cohort of CHD patients or the appropriate waiting period before proceeding with surgery. Patients may have to wait until full resolution of pulmonary tuberculosis before surgery is conducted. We present a case of a 20 year old female presenting with pulmonary tuberculosis complicating Tetralogy of Fallot who successfully underwent surgery after 2 months of intensive phase TB therapy with Isoniazid, Rifampicin, Pyrazinamide and Ethambutol.

Keywords: Cardiac surgery, congenital heart disease (CHD), pulmonary tuberculosis (PTB), Tetralogy of Fallot (ToF).

1. CASE SUMMARY

Presenting illness and physical examination

A 20 year old nulliparous female from Murang’a County, Central Kenya presented to us with history of cough, difficulty in breathing and weight loss for 6 months. The cough was productive of small amounts of clear sputum and was not associated with chest pain. She reported that her cough started after she received COVID-19 vaccination (Moderna). The cough was worse when in a recumbent position and was relieved by sitting up in bed. There was minimal shortness of breath on exertion. Progressively she began noting swelling of both feet. She didn’t report history of night sweats or fever. This was against a background history of recurrent hospital admissions in various local hospitals where she had been treated for unspecified recurrent respiratory tract infection. She reported having been on several courses of antibiotics. Her current
medications included propranolol 20mg once daily and furosemide 20mg once daily. She was a lifetime non smoker and had no history of alcohol or other substance use. Her family history was unremarkable with no history of premature coronary artery disease, sudden cardiac death or congenital heart diseases.

Clinically she was in extremis, cachexic, with cyanosis and profound hypoxia (SPO2 75%) in room air. She had mild pitting bipedal edema at the ankle level. She was not pale. Her pulse rate was 90b/min and blood pressure was 100/70 mmhg. She was afebrile with a temperature of 36.5 degrees Celsius. Her cardiovascular examination findings included a grade 3/6 end systolic murmur heard at the left upper sternal border with a single S2 sound. S1 was normal. The precordium was normal with no heaves or thrills and the apex beat was palpable at the 5th intercostal space mid clavicular line. Peripheral vascular examination was normal. There was notable lower chest wall in-drawing but no obvious chest dysmorphism. Fine crackles were auscultated on the lower chest bilaterally. Abdominal examination was unremarkable with no organomegally or ascites. The rest of the systemic examination was normal.

**Diagnostic workup**

Her basic work up included a complete blood count which showed a white cell count of 6.59 x 10^3 (3.5 -10.5) a raised Hemoglobin 20.0g/dl (12.0-15.5g/dl) which is consistent with a chronic hypoxic state. Her platelet count was normal 219 x 10^3 (150-450). Renal function was normal with a serum creatinine of 65mmol/l (50-120mmol/l). SARs-COV2 PCR test was done using nasal pharyngeal swab and was negative. A HIV test was also negative. A chest X-ray revealed nodular opacities suggestive of pulmonary tuberculosis. This finding was further supported by a computed tomography (CT) scan of the chest and confirmed by positive sputum gene X-pert testing. Transthoracic echocardiography demonstrated a 9mm inlet ventricular septal defect with right-to-left shunting at a gradient of 40mmhg and severe pulmonary stenosis, confirmed by a pulmonary valve maximum pressure gradient of 68mmHg and a continuous wave Doppler velocity of 4.1m/s. There was also hypertrophy of the right ventricle with reduced systolic function. Left ventricular function was preserved with ejection fraction of 57%. Situs was solitus with levocardia position, concordant arterioventricular, ventriculoatrial and atriovenous connections. Electrocardiogram showed R/S ratio >1 in lead V1 and precordial T wave inversion in anterior leads consistent with right ventricle strain.

**Fig 1:** Pre-operative trans-pulmonary echo Doppler measurement showing elevated pressure gradient across the pulmonic valve consistent with pulmonary stenosis.

**Fig 2:** 12 lead ECG showing R/S ratio >1 in lead V1 and precordial T wave inversion in anterior leads consistent with RV strain
Fig 3: selected coronal and transverse CT Scan images of the chest before initiating TB treatment; images show extensive right lung opacities due to Tuberculosis

Management and follow-up

The patient was diagnosed with pulmonary tuberculosis complicating Tetralogy of Fallot and was subsequently admitted to our Intensive Care Unit. She received supplemental oxygen, intravenous diuretics (I.V Furosemide 40mg twice daily) and inotropic support for RV failure with I.V Dobutamine 0.5mcg/kg/min. She was also started on 3 tablets once daily of intensive fixed dose combination anti- TB therapy with Rifampicin 150mg, Isoniazid 75mg, Pyrazinamide 400mg, and Ethambutol 275mg and survived to discharge 14 days later. She was discharged on oral lasix 40mg once daily and continued the oral TB medication. Additionally, she received nutritional support with Ready-To-Use Therapeutic Feeds (RUTF) per local Ministry of Health guidelines. During her review 6 weeks later, she reported significantly less dypnoea and cough. A sputum microscopy with Ziehl Neelsen staining was done and turned negative for Acid Fast Bacilli (negative TB test). Following a multi-disciplinary discussion, a decision was made to proceed with surgery. She was therefore admitted and underwent successful VSD closure with autologous pericardial patch and pulmonary valvotomy, resulting in favorable clinical outcomes. The patient’s oxygen saturation rose to 94% with full resolution of cyanosis and pulmonary valve pressure gradient dropped to 36 mmhg in the first week post surgery.

It has now been 9 months since the patient started TB therapy (completed 3 months ago) and 7 months since surgery. Clinical improvement has been remarkable with full resolution of symptoms (cough and dyspnoea), has gained >10kg and is now able to engage in farming and small scale trading.

2. DISCUSSION

Tetralogy of Fallot (TOF) is a relatively common congenital heart defect that affects the structure and function of the heart. It is characterized by a combination of four cardiac abnormalities: a ventricular septal defect (VSD), overriding aorta, pulmonary stenosis (PS), and right ventricular hypertrophy (RVH). The global prevalence of TOF is estimated to be around 3-7 cases per 10,000 live births. It is observed in various populations and ethnic groups worldwide, without significant geographical variations. TOF is typically diagnosed during infancy or early childhood. However, diagnostic modalities are not easily accessible in Sub Sahara Africa and diagnosis of TOF and other CHDs may be delayed into adolescence and early adulthood (Ansong, 2021) with this case being a succinct example. Patients with uncorrected TOF who survived to late adulthood, like is the case with our patient, often face long-term complications from prolonged cyanosis, including polycythemia, coagulopathy, intracranial abscess, stroke, hyperuricemia, recurrent respiratory infections including pulmonary tuberculosis and neurodevelopemental delay. Surgical intervention is generally recommended in early childhood and even during infancy for TOF (Brown MD, 2005). Patients with unrepaired TOF are also at high risk of infective endocarditis, brain abscess, pulmonary tuberculosis and arrhythmias during their adolescence and adulthood. Cardiac repair in this age group could still be performed with favorable surgical results and long-term outcomes (Yang, 2012).

The finding of pulmonary tuberculosis in our patient (with cyanotic heart disease) is a rare occurrence as reported by Van der Merwe et al who reported that patients with acyanotic CHD (increased or normal pulmonary blood flow) are more

susceptible to develop pulmonary TB than those with cyanotic CHD. Van der Merwe et al explained this situation as reduced pulmonary blood flow and cyanosis in patients with cyanotic-CHD can inhibit growth of Mycobacterium tuberculosis whereas increased pulmonary blood flow and normal pulmonary arterial saturation in patients with acyanotic-CHD can provide a suitable environment to growth (Van der Merwe PL, 1995). In a retrospective study of over 6 years Van der PL et al found that cardiac surgery had to be postponed as a result of pulmonary tuberculosis in 7.2% of all patients in whom it was required. Over the 6-year period of the study, cardiac surgery had to be delayed in 60% of cases with pulmonary tuberculosis and congenital heart lesions so anti-tuberculosis therapy could be completed (Van der Merwe PL, 1995)3. In our case surgery was done after 2 months of TB therapy. The decision was individualized based on good clinical response to TB therapy and the consideration of the urgency of the surgery.

3. CONCLUSION
The occurrence of recurrent respiratory infections in the setting of congenital heart disease (CHD) should prompt intensive case finding for pulmonary tuberculosis using multi-modal imaging and laboratory investigations. The optimal timing of cardiac surgery for patients with TOF and other CHDs complicated with pulmonary tuberculosis warrants further studies on a larger scale in order to come up with algorithmic guidelines. However, we conclude that a 2-month intensive, weight and age adjusted fixed dose combination anti TB regimen with Rifampicin, Isoniazid, Pyrazinamide and Ethambutol is effective in controlling TB infection and minimizing perioperative complications in patients with ToF and concomitant TB infection. The decision regarding the timing of cardiac surgery should be made on an individual basis, considering the patient’s overall clinical condition, response to anti-TB therapy, and the necessity of cardiac intervention and should be done within a multidisciplinary context.

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INFORMED CONSENT
Written informed consent was obtained from the patient to publish this case report, including the images.

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REFERENCES