Double Coronary Artery Fistula from Left Circumflex Artery Draining to the Left Atrium in a Rheumatic Heart Disease Patient – A Case Report

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ABSTRACT

Introduction: Coronary artery fistula (CAF) is a connection between one or more of the coronary arteries and a cardiac chamber or great vessel. This is a rare defect and occurs in only 0.2% of the population. Most of the cases are congenital but acquired causes are also reported. A CAF may occur as an isolated cardiac defect or with other cardiac diseases such as rheumatic heart disease (RHD). Only a few cases of coexisting CAF and RHD have been reported. Local data reports only 0.69% CAFs associated with congenital malformations of the heart. Only 61 patients among all patients who underwent coronary arteriography in 34 years were reported to have a CAFs. We report a case of severe mitral stenosis (MS) with a double CAF from the left circumflex (LCx) artery draining into the left atrium.

Case: A 46-year old female with RHD with severe MS came in due to progressive dyspnea. The coronary angiogram revealed two fistulous tracts originating from the LCx draining into the left atrium. She underwent mitral valve replacement (MVR) surgery, left atrial plication, and closure of the fistula drainage the left atrium. The postoperative course was uneventful.

Discussion: A CAF is often asymptomatic until the second decade of life. Untreated, this may progress and cause ischemic and heart failure signs and symptoms. The presence of MS caused elevated left atrial pressure which might have prevented the increase in the volume of blood draining from the LCx artery to the left atrium through the fistulas. Hence, the MS might have prevented the dilatation of the two fistulas. Surgical correction is also indicated in the fistulas since resolution of the mitral stenosis with MVR will decrease the LA pressure which might result to dilatation and increased drainage of the fistulas causing complications later.

***Keywords**: Coronary artery fistula, Rheumatic heart disease, Case Report*

INTRODUCTION

A coronary arterial fistula (CAF) is rare defect which connects one or more of the coronary arteries to a cardiac chamber or great vessel. These are found in 0.2% of patients undergoing coronary angiography. They are present in only 0.002% of all patients with congenital heart disease. Three consecutive studies involving all patients who underwent coronary angiogram in the Philippine Heart Center from year 1975 to 2009 documented only 61 cases of coronary artery fistulas. CAF may either be congenital (64%) or acquired (36%). They commonly originate from the right coronary artery and terminate in the right side of the heart in over 90% of cases. Multiple fistulas are even rarer in occurrence. CAF may be found coexisting with other cardiac disease such as rheumatic heart disease (RHD) in a few reported cases.

CASE

A 46-year old female, married, Filipino, from Davao City came in due to progressive dyspnea.

Six years prior to admission (PTA), patient experienced undocumented fever, chills, and cough. Consult was done and she was managed as pneumonia. She had no other symptoms. A grade 3/5 diastolic murmur heard best at the apex was noted on physical examination. 2d-echocardiography (2D echo) was done which revealed RHD with severe MS. Surgery was advised but with no consent. She was lost to follow up. Four years PTA, the patient had episodes of palpitations and on and off bipedal edema. She still had good functional capacity that time.

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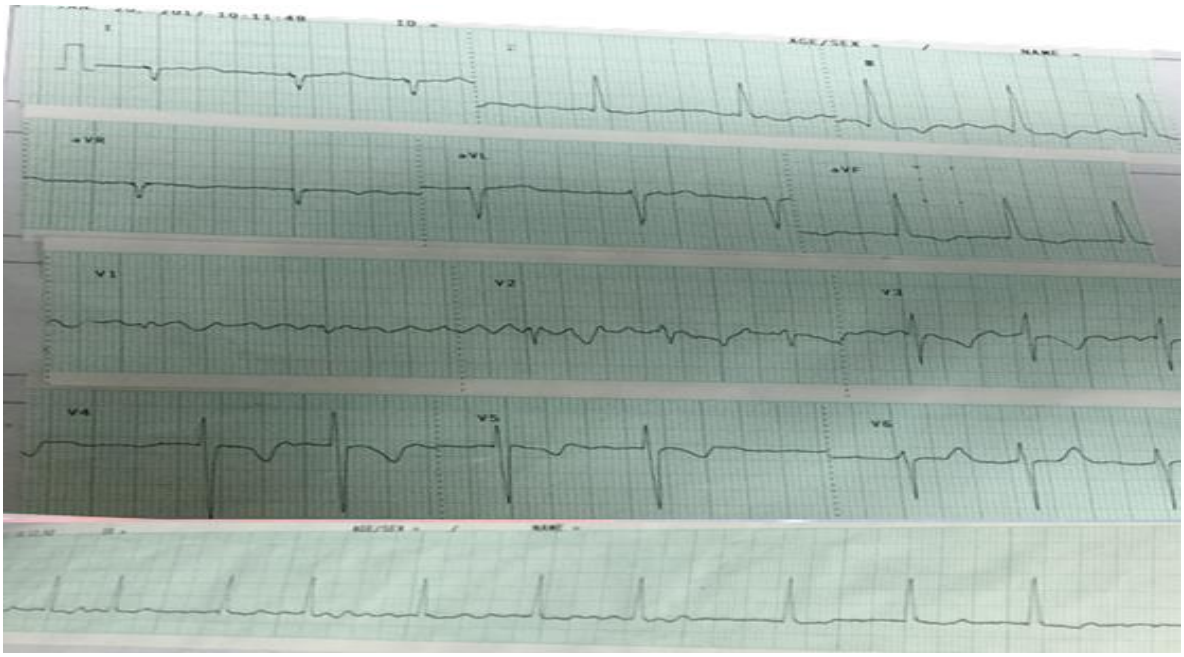

Figure 1. Pre-operative ECG: Atrial fibrillation in moderate ventricular response, with right axis deviation, and right ventricular hypertrophy with strain pattern.

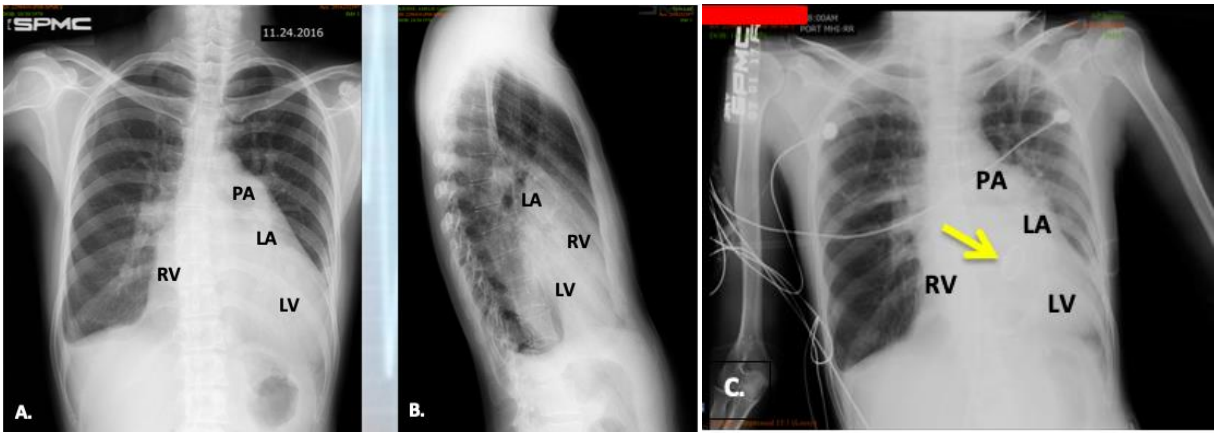

Figure 2. Chest radiography. (Preoperative) A. Postero-anterior view showing biventricular cardiomegaly and left atrial enlargement with blunting of bilateral costophrenic sulci. B. Lateral view showing obliteration of retrosternal and retrocardiac space. (Post-operative) C. AP view revealed biventricular and left atrial cardiomegaly that are regressing in size and bilateral minimal pleural effusion. Mechanical mitral valve (yellow arrow) can also be identified. PA: pulmonary artery; LA: left atrium; LV: left ventricle; RV: right ventricle.

Two years PTA, there was persistence of palpitations. She developed exertional dyspnea, easy fatigability, two-pillow orthopnea, and progression of bipedal edema. She was admitted at the intensive care unit and had an episode of generalized tonic-clonic seizure and aphasia for three days. She was managed as cardioembolic stroke and congestive heart failure (CHF) secondary to RHD with severe MS (0.8 cm^2) and thrombus formation. Valve replacement surgery was again offered without consent.

Patient had progressive dyspnea and easy fatigability this time affecting some of her daily task. She finally consented for surgery and was admitted.

She had a prior history of severe tonsillitis at 9 years of age which required hospitalization. She is a non-smoker and occasional alcoholic beverage drinker. Bronchial asthma on the maternal side and coronary artery disease on the paternal side are present in the family history. One brother died of sudden cardiac death at the age of 43.

She was examined awake, oriented, with stable vital signs. Blood pressure were 100/70 mmHg on both upper extremities and 100/60 mmHg on both lower extremities. Body mass index was 16.2 kg/m^2.

The pertinent physical findings centered on the cardiac findings. Cardiac examination revealed dynamic

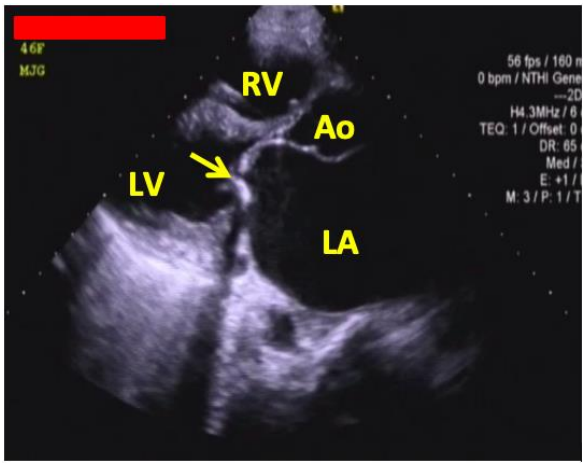

Figure 3. Pre-operative 2D-echocardiography showing severe mitral stenosis (yellow arrow) with area of 0.7 cm^2 and dilated LA with no thrombus formation. LA = Left Atrium, LV = Left Ventricle, RV = Right Ventricle, Ao = Aorta

precordium with point of maximal impulse (PMI) at sixth intercostal space (ICS) left anterior axillary line. Heave was appreciated at the 5th ICS left parasternal area. No thrill was palpated. The cardiac rhythm was irregularly irregular. Soft S1 and loud S2 were heard. There was note of a grade 2/6 diastolic rumbling murmur heard best at 6th ICS at the left anterior axillary line and grade 3/6 holosystolic murmur at the 5th and 6th ICS, left parasternal border with Carvallo's sign. No opening snap was appreciated. Decreased breath sound were noted at the bi-basal lung fields.

The patient was admitted as a case of RHD with MS, TR, (+) left atrial dilation, (-) left ventricular dysfunction with ejection fraction (EF) of >55%, atrial fibrillation (AF) in moderate ventricular response (MVR) functional class III.

Her complete blood count, electrolytes, and bleeding parameters were all unremarkable (Appendix A). ECG showed AF in MVR with right axis deviation (RAD), and right ventricular hypertrophy (RVH) with strain pattern.

Preoperative chest X-ray PA/L view showed biventricular and left atrial cardiomegaly with bilateral minimal pleural effusion (*Figure 2A and 2B*).

2D echo (*Figure 3*) revealed severe MS, mild mitral regurgitation (MR), mild aortic regurgitation (AR), and moderate TR with moderate pulmonary hypertension with EF at >55%. Left atrium was dilated with no thrombus formation. All the other chambers were normal.

The coronary angiogram showed angiographically normal coronary arteries but with incidental finding of two fistulous tracts originating from the proximal left circumflex artery draining into the left atrium (*Figure 4*).

The patient underwent MVR using mechanical valve (SJM 27mm mechanical) and closure of the terminal site of the two fistulous tracts at the left atrium (*Figure 5*). Left atrial plication and IOTEE were also done.

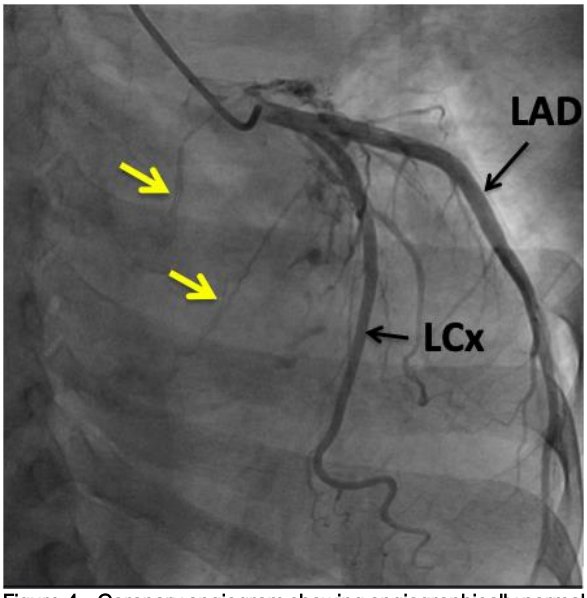

Figure 4. Coronary angiogram showing angiographically normal coronary arteries with two fistulous tracts (yellow arrow) from the proximal left circumflex artery draining into the left atrium. LAD = Left anterior descending artery, LCx = Left circumflex artery

Post-operatively, there was significant improvement in the symptoms of the patient. Post-operative chest radiography (*Figure 2C*) revealed biventricular and left atrial cardiomegaly that are regressing in size and bilateral minimal pleural effusion.

Repeat 2D-echo (*Figure 6*) done post operatively showed metallic prosthetic valve at the mitral position with adequate opening and closing motion.

The final diagnosis was RHD with MS severe, MR mild, TR moderate, aortic regurgitation (AR) mild, CAF from LCx to left atrium, (+) dilated left atrium, AF in MVR, ejection fraction >55 %, functional class III, status post mitral valve replacement with ligation of the fistula, left atrium plication, and IOTEE.

The patient was discharged improved with regular follow up. Her maintenance medications include warfarin 2.5 mg/tab 1 tab once a day, spironolactone 25mg/tab 1 tab once a day, and carvedilol 6.25 mg/tab 1 tab 2x a day. She is back to her functional capacity.

DISCUSSION

CAF is an abnormal connection between one or more of the coronary arteries and a cardiac chamber or a great vessel. It bypasses the myocardial capillary bed.[1] CAFs may either be congenital (64%) or acquired (36%).[2,3]

CAFs are found in 0.2% of patients undergoing coronary angiography. It is present in 0.002% of all patients with congenital heart disease.[4] The largest case series of CAF is reported by the Cleveland Clinic, which found 225 patients with incidental CAF out of 126,595 coronary catheterizations (incidence of 0.18%), performed during a span of 28 years.[5] A 10-year local study among patients

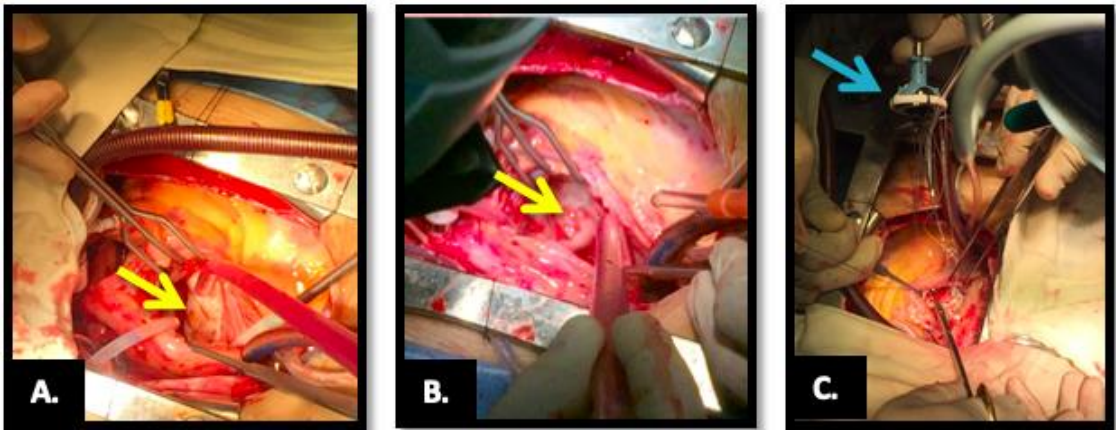

Figure 5. Intraoperative view. A. First fistulous tract (yellow arrow). B. Second fistulous tract (yellow arrow). C. Mitral valve replacement was also done using SJM 27mm mechanical (blue arrow).

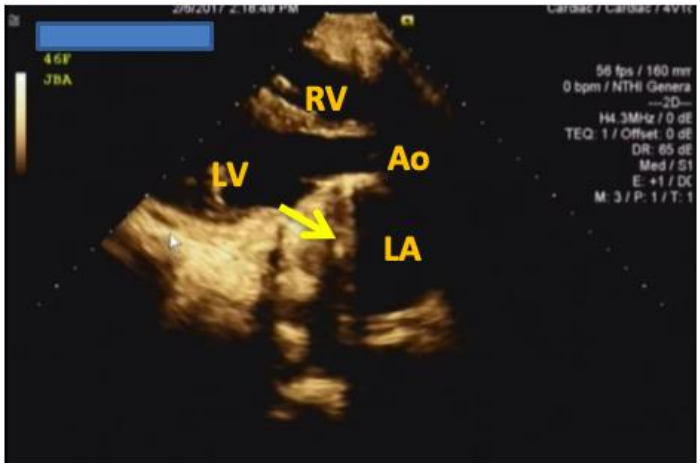

Figure 6. Post-operative 2D echocardiography: Post-operative echo showed metallic prosthetic valve at the mitral position with adequate opening and closing motion. Metallic valve shadow can be appreciated in the echo. LA = Left Atrium, LV = Left Ventricle, RV = Right Ventricle, Ao = Aorta.

who underwent coronary artery disease (CAD) shows that there are only 0.69% congenital malformations of the heart, only 26% have CAF, and 9% has coronary-cameral fistula.[6] Three studies conducted at the Philippine Heart Center from 1975 to 2009 show that only 61 patients were found to have CAF among those who underwent cardiac catheterization.[7-9] Multiple CAF are an even rarer entity and only a handful of cases have been reported in the literature to date.[10,11]

Fifty-two percent of the CAFs originate from the right coronary artery (RCA), 30% are from the left anterior descending coronary artery (LAD), 18% of cases originate from the left circumflex coronary artery (LCx), and 5% from multiple vessels.[12] The right side of the heart is the most common point of termination. It encompasses over 90% of the cases and rarely will it drain to the left side of the heart.[13] Another study report contrasting results. 70% of the fistula originated from the left coronary artery (LCA) while only 26% were from the RCA. However, among those that originated from the LCA, only four (9%) were from the LCx.

Fistula originating from LCx and are draining to the left atrium are rare forms of CAF. Furthermore, double fistula with this origin and drainage are rarer cases of CAF. Only one other case of two fistulous tracts from LCx draining into the LA is reported in literature. This was the case of a 62-year old female in India which had two CAF from distal LCx draining into left atrium.[14]

Most CAFs are small and do not cause any symptoms. It is only when a fistula reaches two times a coronary size that signs and symptoms develop.[15] The feeding artery of the CAFs may drain from a main coronary artery or one of its branches. It is usually a dilated and tortuous artery terminating in one of the cardiac chambers or a vessel.[16] It is vital to recognize the anatomic arrangement of these fistulae because some origin and course of the fistula predispose the patient to the development of myocardial ischemia, heart failure, arrhythmia, or sudden death.[17,18] hence, requiring the need for further treatment and surgical intervention.[19] The physiologic consequences of CAFs depend on the volume of blood flowing through them, the chamber, or vascular bed into which they drain, and the myocardial ischemia that results from a coronary steal.[20,21,22] A steal occurs when blood flow is shifted away from the distal coronary vascular bed.[23] Our patient did not present with this steal phenomenon since her fistula were small, non-tortuous, and non-dilated. Hence, her LV contractility and ejection fraction in 2D-echo were normal.

The most common physical examination finding is an asymptomatic continuous murmur, loudest over the

precordium, often mistaken as a patent ductus arteriosus.[24,25,26] The murmur is heard over the mid-chest or even lower, rather than below the left clavicle and typically peaks in mid to late diastole. If the fistula connects to the left ventricle, only an early diastolic murmur may be heard.[27] Our patient had a grade 2/6 diastolic murmur heard best at sixth ICS at the left anterior axillary line and a grade 3/6 holosystolic murmur at the 5th and 6th ICS with Carvallo's sign indicative of mitral stenosis and tricuspid regurgitation respectively.

Congestive heart failure (CHF) signs and symptoms such as exertional dyspnea, easy fatigability, bipedal edema, and orthopnea, similar to what our patient had, can be attributed to any functional or structural cardiac disorder that impairs the ventricle's ability to fill with or eject blood. The most common causes are ischemic heart disease, idiopathic dilated cardiomyopathy, hypertension, and valvular heart disease.[28] Our patient had no predisposing factors for the three leading causes. Furthermore, the murmur on physical examination point to a common valvular heart disease (VHD) – mitral stenosis.

The coexistence of RHD and CAF is another interesting facet of this case. Patients for valve replacement oftentimes undergo coronary angiography where CAF, when present, is usually incidentally diagnosed. Only a few cases of CAFs with coexistent rheumatic heart disease have been reported globally.[29,30] However, RHD is prevalent in the Philippines, hence a higher incidence (22%) of coexistent CAF and RHD was reported in some studies.[7,8,9]

The mitral valve is the most affected valve in RHD.[31,32] Severe MS occurs when the mitral valve opening is reduced to <1.5 cm^2. An LA pressure of ~25 mmHg is then required to maintain a normal cardiac output (CO).[28] This elevated LA pressure causes passive backward transmission to the lungs resulting to pulmonary hypertension. Severe pulmonary hypertension results in RV enlargement, secondary TR, and pulmonic regurgitation (PR), as well as right-sided heart failure.[33]

Severe MS may also present with heart failure signs and symptoms. As MS progresses, patient develops progressive dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. The development of persistent AF is generally associated with acceleration of symptoms progression.[33]

The presence of the MS in our patient may have delayed the progression in sizes of these fistula. An elevated LA pressure is required to maintain a normal cardiac output in patients with MS. The high pressure at the LA may have prevented the increase in volume that would have drained from the LCx to the LA through the fistula. This may have prevented the dilatation and tortuosity of the fistulas which would have occurred in an otherwise normally structured heart. The two fistula in our patient might not have caused nor contributed in her heart failure signs and symptoms. Rather, the failure symptoms are due to the severe MS.

The main diagnostic techniques to detect CAFs are cardiac catheterization and angiography.[34] Initial diagnostic catheterization is needed to assess the hemodynamic significance of the fistula and to provide detailed anatomy of the fistula, particularly, the size, origin, course, presence of any stenosis and the drainage site.[35] The electrocardiogram and chest x-ray may show unremarkable results. Two-dimensional and color Doppler echocardiography are helpful in demonstrating dilation of the affected coronary artery. Color flow mapping may show the site of drainage but the detailed anatomy may be difficult to define.[36] Magnetic resonance imaging may also help in confirming the diagnosis.[37]

The goal of treatment is the occlusion of the fistula and preserving the normal coronary blood flow.[38] The usual indications for treatment include the presence of a large or increasing left-to-right shunt, left ventricular volume overload, myocardial ischemia, left ventricular dysfunction, congestive cardiac failure, and for prevention of endocarditis/endarteritis. The treatment options for coronary arterial fistulas include surgery or catheter closure.[39] Complete occlusion of the fistula may be achieved in >95% of cases after surgery although the exact incidence of residual fistulas is not known. The mortality in the repair of CAFs is from 0% to 4%.[40]

Our patient has a giant left atrium measuring >6 cm.[41] The main indication for left atrial volume reduction is the presence of intracardiac or extracardiac compressive symptoms such as arrhythmia which our patient has. The rheumatic process causes strain and loss of tone on the elastic fibers of the tissue resulting in irreversible left atrial enlargement. Reduction of the left atrial size together with replacement of the mitral valve reduces the pressure effect with favorable effect on the postoperative course.[38]

Surgery was the best option for our patient. She underwent MVR using SJM 27mm mechanical valve, left atrial plication, and closure of the terminal sites of the two fistulous tracts into the left atrium. MVR may decrease the LA pressure with subsequent increase in the volume draining through the fistulas resulting to increase in size and tortuosity of the fistulas. This may later cause complications of ischemia or recurrence of heart failure signs. Thus, there was a need to ligate the "asymptomatic" fistula together with the MVR. The patient was discharged improved with warfarin. She has regular follow-up for protime and INR monitoring at the out-patient department.

SUMMARY

A 46-year old female from Davao City came in with progressing signs and symptoms of heart failure eventually leading to a diagnosis of rheumatic heart disease. Coronary angiogram revealed an incidental finding of two fistulous tracts from left circumflex artery draining to the left atrium. Coronary artery fistula itself is a rare abnormality. Fistula from the LCx draining to left atrium is relatively less common compared to other forms of coronary artery fistula. The coexistence of the CAF with RHD is rare with only few cases reported in literature.

The patient presented with heart failure signs such as easy fatigability, exertional dyspnea, bipedal edema, and

orthopnea. These symptoms are commonly seen in a patient with severe MS. Though these signs and symptoms can also result from a large CAF, two small non-tortuous CAFs did not cause or contributed to the clinical presentation of the patient. The presence of the mitral stenosis caused elevated LA pressure which might have delayed or even prevented the progression in the size of the fistulas draining into the LA. Hence, if only mitral valve replacement was done, resolution of mitral stenosis will decrease the LA pressure and allow increase in volume draining from the LCx to the LA through the fistulas. This may later cause complication which might prompt a repeat surgery to repair the fistulas. Hence, mitral valve replacement, left atrial plication with closure of the two fistulas was done in the patient.

Post-operatively, there was a remarkable improvement in the patient's signs and symptoms with resolution of her heart failure signs.

CONCLUSION

CAF is a rare cardiac abnormality. Furthermore, cases of CAF in association with RHD are even rarer. Clinical manifestations, treatment, and prognosis of CAF and RHD depend on the origin and drainage of the anomalous tract and the valvular involvement. Most CAFs are incidental findings for other cardiac diseases. Meticulous work up including coronary angiogram and hemodynamic studies should be done to diagnose, describe, and elucidate the interplay of the CAFs with the other cardiac problems. The sequelae of RHD leading to severe MS and left atrial enlargement with the presence of two fistulous tracts present major challenges both pre- and post-operatively. Mastery of the pathophysiology of the heart and its vessels is paramount in the management of these cases. Sometimes the management given to our patients may awaken new problems with deleterious consequences in the future.

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APPENDIX A

Laboratory Test Results of the Patient

COMPLETE BLOOD COUNT		
Parameters	**Results**	**Reference Ranges**
Hemoglobin	124 g/L	115-155 g/L
Hematocrit	0.37	0.36-0.48
RBC Count	4.4 x 10^6/uL	4.20-6.10 x 10^6/uL
WBC Count	8.54 x 10^3/uL	5.0-10.0 x 10^3/uL
Differential Count		
Neutrophil	76 %	55-75%
Lymphocyte	15 %	20-35%
Monocyte	5 %	2-10%
Eosinophil	3 %	1-8%
Basophil	1 %	0-1%
Platelet Count	216 x 10^3/uL	150-400 x 10^3/uL

BLOOD CHEMISTRY		
Fasting Blood Glucose	5.15 mmol/L	4.10-6.60 mmol/L
Creatinine	62.39 umol/L	39.0-113.0 umol/L
Blood Urea Nitrogen	3.72 mmol/L	2.9-7.1 mmol/L
Albumin	35.5 g/L	35.0-50.0 g/L
SGPT	15.15 U/L	14-63 U/L
SGOT	29.9 U/L	15-41 U/L
Sodium	138.96 mmol/L	136-144 mmol/L
Potassium	3.6 mmol/L	3.6-5.1 mmol/L
Magnesium	0.82 mmol/L	0.74-1.03 mmol/L
Calcium	2.15 mmol/L	1.75-2.39 mmol/L
Chloride	103.8 mmol/L	101-111 mmol/L

COAGULATION STUDIES	
Prothrombin Time	Patient: 19 seconds Control: 14.2 seconds INR: 1.63 % Activity: 50 %
Activated Partial Thromboplastin Time	Patient: 34.3 seconds Control: 32.5 seconds

THYROID HORMONES		
Parameters	**Results**	**Reference Ranges**
Free Thyroxine	14.07 pmol/L	7.50-21.10 pmol/L
Thyroid Stimulating Hormone	1.34 uIU mL	0.34-5.60 uIU mL