Loud and Clear: A Case Report on Ortner's Syndrome (Cardiovocal Syndrome)

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Abstract

Introduction: Ortner's syndrome encompasses any intrathoracic process resulting to stretching of the recurrent laryngeal nerve. The stress to this nerve weakens the intrinsic muscles of the larynx and manifests as vocal hoarseness.

Case: A 46-year-old female was admitted due to progressive hoarseness. She tolerated this for six months until the onset of other symptoms like orthopnea, bipedal edema, and chest pain. She consulted various doctors and was initially diagnosed with chronic laryngitis from gastroesophageal reflux disease. She was given omeprazole and spironolactone that afforded little help.

On examination, the precordium was dynamic with heaves and thrills, PMI was displaced, and systolic murmurs were heard. The ECG revealed left ventricular hypertrophy with left atrial abnormalities. Chest radiograph showed left-sided cardiomegaly. A 2D-echocardiogram divulged severe mitral regurgitation, anterior mitral valve leaflet thickening, and dilated left atrium. Therefore, the impression was Ortner's syndrome, rheumatic heart disease.

The patient underwent mitral valve replacement. She was given warfarin, bisoprolol, losartan, spironolactone, and vitamin B complex. At subsequent visits, she reported improvement in her voice. Penicillin injections were also given as secondary prophylaxis. A repeat 2D-echocardiogram displayed a decrease in size of the left atrium.

Conclusion: This case has emphasized three important points: First, a comprehensive history and physical examination is vital to reach the correct diagnosis. Second, once the common causes of hoarseness have been excluded yet symptoms persist, it is therefore prudent to look beyond the larynx. And third, the vocal cord paralysis experienced by the patient is just the tip of the iceberg. Her heart is the one dying to be heard.

Keywords: ortner's syndrome, cardiovocal syndrome, recurrent laryngeal nerve paralysis, vocal cord paralysis, hoarseness, heart failure

Introduction

Have you even wondered how the human voice works? Voice is produced through the steady airflow from the lungs that travels into the larynx. The muscles in the larynx control the tension of the vocal cords, resulting to periodic opening and closing of the vocal tract.[1] Once in motion, the vocal cords effectively convert this steady airflow into a series of audible impulses that are further filtered and articulated by the tongue, palate, cheeks, and lips to create highly intricate array of sounds.[1-2] Any pathology along this sequence can result to change in voice.

The value of our voice is often unrecognized until it is damaged or lost. Hoarseness is the general term to describe abnormal voice change. The voice may sound breathy, raspy, or strained. And this can be the dominant symptom in a patient with a severe heart disease, like in this case.

This care report aims to: (1) present a case of a 46-year old female who came in due to hoarseness; (2) discuss laboratory results, management, and the outcome of this case; and (3) discuss Ortner's syndrome - its epidemiology, pathophysiology, clinical features, management, and prognosis.

Case

A 46-year-old female, married, Filipino, working as a janitor was admitted for hoarseness. Nine months prior to admission (PTA), the patient noted that her voice was progressively becoming breathy and croaky. This was associated with occasional dyspnea. She tolerated her condition until three months PTA, the change in her voice was already accompanied by two-pillow orthopnea and grade one bipedal edema. There were no symptoms of

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paroxysmal nocturnal dyspnea, dysphagia, hemiparesis, and tremors. She decided to consult an otolaryngologist and was initially diagnosed with chronic laryngitis secondary to gastroesophageal reflux disease. She was given omeprazole and spironolactone that afforded little help. Ancillary diagnostics such as cervical computed tomography turned out to be unremarkable.

A month PTA, the patient complained of worsening hoarseness that makes speaking impossible without having to strain. Furthermore, symptoms of palpitations and non-productive cough were notable. There were few episodes retrosternal chest pain described as dull, non-radiating, and with a pain scale of 5/10. With the worsening of symptoms and marked limitation of physical activity, the patient was then advised admission.

The patient was a G4P4(4004) mother, who delivered vaginally. She was a non-hypertensive, non-diabetic, and non-asthmatic. She had no known food and drug allergies and denied alcohol and illicit drug abuse. She had no history of mania and depression.

On physical examination, the patient was awake, conscious, oriented, and in mild respiratory distress. Vital signs were as follows: blood pressure of 90/60 mmHg on all extremities, heart rate of 70 beats per minute, respiration rate of 28 cycles per minute, and axillary temperature of 36 degree Celsius. The patient had a body mass index of 18 kg/m^2. Dental carries in teeth no.17, 27, 28, 35, 36, 37, 45, 46, and 48 were detected. Weakness of the left vocal cord was demonstrated through direct laryngoscopy (Figure 1). The chest expanded equally with clear breath sounds. The precordium was dynamic with heaves and thrills palpated on the left parasternal border fifth intercostal space. The point of maximal impulse was at the fifth intercostal space - left axillary line. There was a grade 4/6 systolic murmur at the fifth intercostal space of the left parasternal border, and a grade 3/6 holosystolic murmur at the apex radiating to the axilla. No tumor plop was heard. The abdomen was flat with normoactive bowel sounds. It was soft and non-tender on palpation, and negative for fluid wave test. The extremities had 2+ pulses on all test points. Grade one bipedal edema was detected.

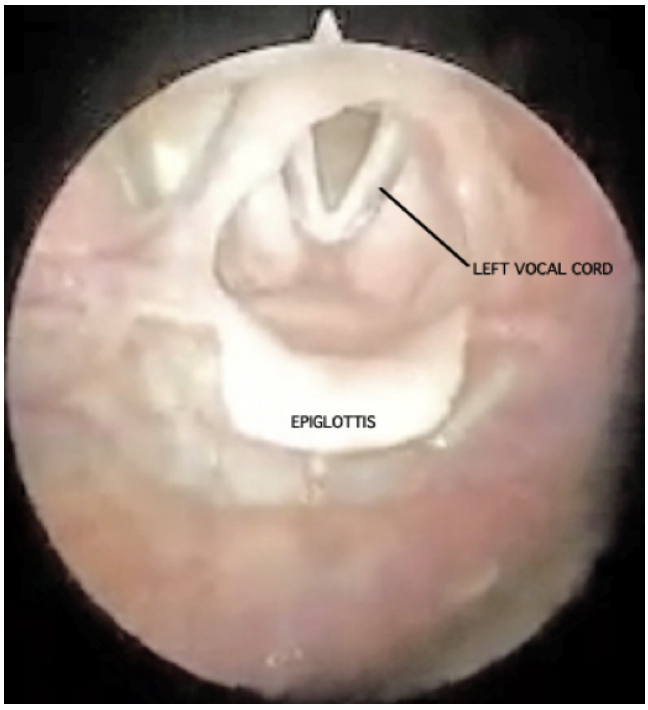

Figure 1. Direct laryngoscopy. This shows the weakness of the left vocal cord. The vocal cords were non-hyperemic and no mass was seen.

The patient was able to recall recent and remote memory. The funduscopic examination was unremarkable. The KLM test was normal. The timed swallowing test was less than 15 seconds. The palatal arch rose symmetrically while the uvula stayed at midline. A bilateral gag reflex was elicited. There was no evidence of tongue atrophy nor fasciculations.

A 12-lead electrocardiogram revealed sinus bradycardia, left ventricular hypertrophy by Cornell criteria, and left atrial abnormalities. The chest radiograph (Figure 2) showed left-sided cardiomegaly with mild pulmonary congestion. A transthoracic echocardiogram divulged severe mitral stenosis with mitral valve area of 0.65 cm^2 by planimetry and 0.83 cm^2 by pressure half time with peak gradient of 27 mmHg and mean gradient of 15 mmHg, severe mitral regurgitation, moderate tricuspid regurgitation, normal left ventricular dimension with normal systolic function, normal right ventricular dimension with good contractility, dilated left atrium, moderate pulmonary hypertension, and left

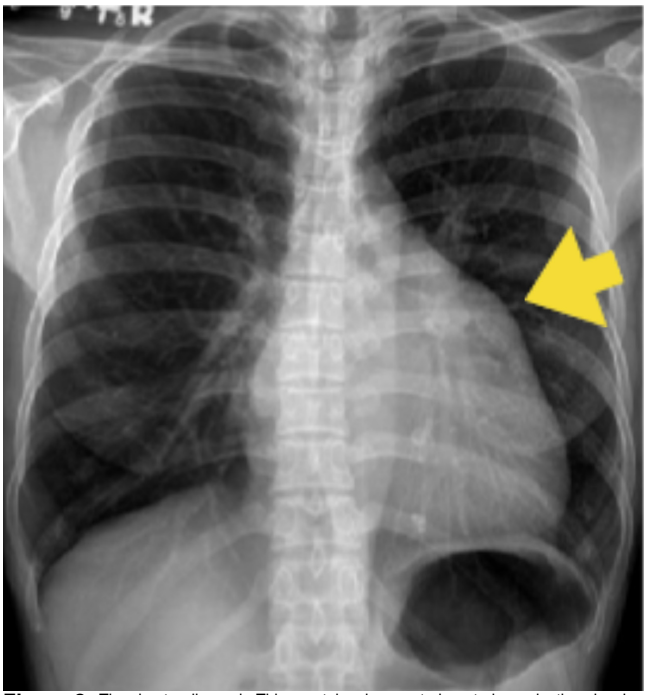

Figure 2. The chest radiograph. This was taken in a posterio-anterior projection showing that the pulmonary vascular markings are prominent with the heart that is enlarged with left ventricular prominence and bulging of the left atrial appendage (yellow arrow). The cardiothoracic ratio is 0.54.

ventricular ejection fraction of 69%. A thickened anterior mitral valve leaflet with diastolic doming motion was also visualized.

Thus, the diagnoses to this case were Ortner's syndrome, heart failure with preserved ejection fraction, rheumatic heart disease (RHD), severe mitral stenosis, severe mitral regurgitation, moderate tricuspid regurgitation, dilated left atrium, left ventricular ejection fraction of 69%, sinus rhythm, NYHA functional class III.

The patient had coronary angiography, which showed normal coronary arteries. Dental restoration and extraction was done prior to cardiac surgery. She underwent mitral valve replacement without note of post-operative complications. Upon discharge, she was advised to avoid overcrowded places and limit sodium intake to two to three grams a day. She was given warfarin 2.5 mg tablet daily and titrated to therapeutic level to prevent systemic embolization, thromboembolism, and valve thrombosis. Other medications included bisoprolol 2.5 mg, losartan 50 mg, spironolactone 25 mg, and vitamin B complex tablet. A month after the surgery, the patient subjectively noted regain of some of her voice. A flexible videostroboscopy (Figure 3) was done, which revealed neither vocal cord paralysis nor mass during adduction and abduction exercises. On subsequent out-patient follow-up, the voice continued to improve. She was given benzathine penicillin G regularly as secondary prophylaxis for the rheumatic fever. A repeat transthoracic echocardiogram (Figure 4) showed the presence of a metallic prosthetic valve at the mitral valve position, mitral regurgitation decreased from severe to trivial, tricuspid regurgitation increased from moderate to severe, and the left atrium decreased from 47 mm^2 to 35 mm^2. One year after the surgery, there was significant improvement on the patient's voice.

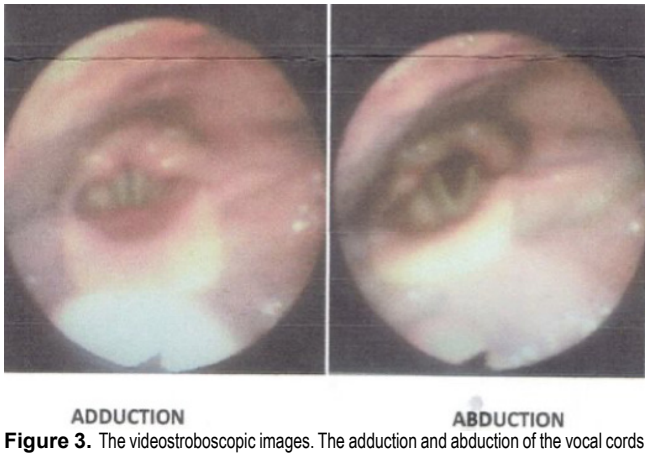

Figure 3. The videostroboscopic images. The adduction and abduction of the vocal cords were normal.

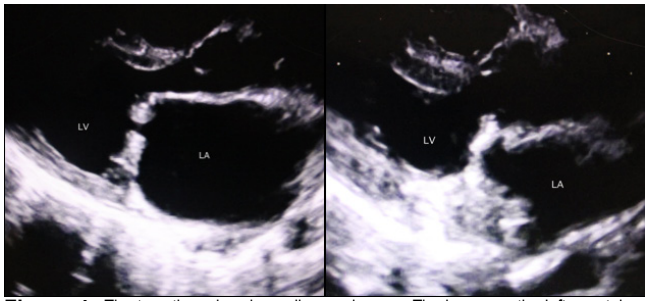

Figure 4. The transthoracic echocardiogram images. The image on the left was taken pre-operatively showing a dilated left atrium (LA). The image on the right showed a decrease in the LA size after surgery.

Discussion

We were presented here a case of a 46-year-old female with a history of worsening hoarseness and symptoms of heart failure. Pertinent in the physical examination were cardiac murmurs and a displaced apex beat signifying cardiac enlargement. Thus, we concluded that the hoarseness was due to recurrent laryngeal nerve paralysis from the compression of an enlarging heart.

The left recurrent laryngeal nerve (Figure 5) branches off the vagus nerve at the arch of the aorta, travels around the lateral aspect of the ligamentum arteriosum, passes up

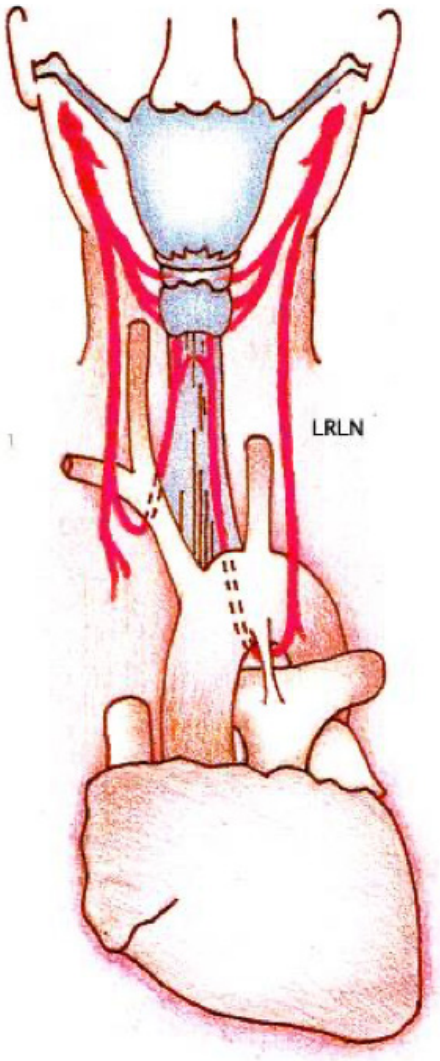

Figure 5. The transthoracic echocardiogram images. The image on the left was taken pre-operatively showing a dilated left atrium (LA). The image on the right showed a decrease in the LA size after surgery.

the right side of the aortic arch, and ascends close to the tracheoesophageal groove before innervating the intrinsic laryngeal muscles. Its long course makes it more vulnerable to trauma, either by compression or stretching, which may result to paralysis of the group of muscles it innervate – the laryngeal muscles that are responsible for voice production.[3]

A rare and/or an under reported clinical entity called Ortner's syndrome or cardiovocal syndrome is defined as recurrent laryngeal nerve paralysis secondary to any intra-thoracic pathology.[4,5,6] It was first described in 1897 by Norbert Ortner in a patient who presented as hoarseness with a concomitant cardiac finding of a dilated left atrium secondary to mitral stenosis, like in our case.[7,8,9] It has now been estimated that the incidence of this syndrome occurring secondary to mitral stenosis is 0.6% to 5% worldwide.[10,11,12] Based on local data, the Philippine Journal of Otolaryngology - Head and Neck Surgery published two cases of cardiovocal syndrome in 2011.[12] In our hospital, this is the first documented case of Ortner's syndrome.

According to various authors, a variety of intra-thoracic abnormalities, such as a dilated pulmonary artery or an aortic aneurysm, could cause trauma to the left recurrent laryngeal nerve.[13,14,15,16,17,18] In this case, the dilated left atrium with a dimension of 47 mm^2 was the primary culprit. In the review of case reports, a left atrial dimension as large as 41 mm^2 was able to cause Ortner's syndrome.[18]

A benign clinical presentation such as change in one's voice is seldom associated with a failing heart. When it becomes symptomatic, heart failure has a poor prognosis. Community-based studies indicate that 60% to 70% of patients die within five years of diagnosis.[19] The functional class could also be used to predict clinical outcome. In a journal that correlates functional class with mortality, a New York Heart Association (NYHA) of III, like our patient, predicts a mortality rate of as high as 34.3%.[20] This underlines the significance of early diagnosis and intervention as NYHA I and II has lower mortality rate by at least 15% and patients who had undergone mitral valve replacement had an improved overall 10-year survival rate of 70%.[21]

Patients with RHD are often asymptomatic for many years before their valvular disease progresses to cause cardiac failure.[22] Its diagnosis is generally confirmed by transthoracic echocardiogram to assess valve morphology and severity of dysfunction. Using the World Heart Federation criteria for the echocardiographic diagnosis of RHD in individuals with no history of acute rheumatic fever, the definite diagnosis of RHD is made for this patient based on the pathologic mitral regurgitation with morphologic features of anterior mitral valve leaflet thickening and restricted leaflet motion.[23]

The patient was advised to avoid overcrowding as primary prevention by elimination of major risk factors for streptococcal infection. Secondary prevention for this patient would entail receiving long-term benzathine penicillin G injections.[22] She was also advised to restrict sodium intake to two to three grams a day. Upon discharge, she was prescribed warfarin, bisoprolol, losartan, spironolactone, and vitamin B complex. According to the American College of Cardiology/American Heart Association, indefinite anticoagulation with a vitamin K antagonist after valve replacement decreases the risk for major systemic embolization, thromboembolism, and valve thrombosis when target INR of 2.5 is reached.[24] Thus, regular monitoring and follow-up is vital for this patient. For the treatment of heart failure with preserved ejection fraction, there is still no single effective treatment achieved in randomized controlled trials. Therapeutic targets in this case include control of congestion, stabilization of heart rate and blood pressure, and efforts at improving exercise tolerance.[25]

The return of laryngeal muscle strength depends on the duration the recurrent laryngeal nerve needs in order to re-myelinate. Therefore, spontaneous recovery can range from hours to months. And in advance nerve damage, the voice improvement may be delayed, or worse, may only be partial despite an impeccable repair of the underlying cardiac lesion.[10] Vitamin B complex may improve motor nerve regeneration following an injury,[26] thus it was given to our patient. The qualitative assessment of the patient's voice or the objective evaluation of the recurrent laryngeal nerve is one of the limitations in this case.

Conclusion

Heart failure is a common debilitating clinical illness. The chance of it presenting as hoarseness is rare. It can mislead the physician to another diagnosis. Hence, it is important to recognize hoarseness secondary to a cardiovascular disease because treatment in this case is different.

This case has emphasized three major points. First, a comprehensive history and physical examination is vital to reach the correct diagnosis. Second, that clinicians must not limit themselves to the most obvious differentials. Once the common causes of hoarseness have been excluded yet symptoms persist, it is therefore prudent to look beyond the larynx. And third, the vocal cord paralysis experienced by the patient is just the tip of the iceberg. Her heart is the one dying to be heard.

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Appendices

The complete laboratory work-up of the patient.

Appendix A. The complete blood count	
Hemoglobin	131 g/L
Hematocrit	0.40
RBC count	4.81 x 10^6/uL
WBC count	9.5 x 10^3/uL
Neutrophils	72 %
Lymphocytes	19 %
Monocytes	5 %
Eosinophils	3 %
Basophils	1 %
Platelet count	262 x 10^3/uL
MCV	82.70 fL
MCH	27.2 pg
MCHC	32.9 g/dL

Appendix B. The urinalysis result	
Physical examination:	
Appearance	Clear
Color	Yellow
Urine chemistry:	
Protein	Negative
pH	5.5
Specific gravity	1.013
Glucose	Negative
Microalbumin	Negative
Urine bilirubin	Negative
Urine urobilinogen	3.4 umol/L
Nitrite	Negative
Leukocyte esterase	Negative
Urine ketone	Negative
Urine flowcytometry:	
RBC	2 /uL
WBC	4 /uL
Epithelial cells	4 /uL
Cast	0 /uL
Bacteria	0 /uL
Crystals:	
Amorphous urates	0 /uL
Calcium oxalates	0 /uL
Uric acid	0 /uL
Amorphous phosphates	0 /uL
Others:	
Mucus threads	1.000 /uL

Appendix C. The clinical chemistry results	
Sodium	139.9 mmol/L
Potassium	3.6 mmol/L
Calcium	2.42 mmol/L
Magnesium	0.77 mmol/L
BUN	5.76 mmol/L
Creatinine	72.0 umol/L
SGPT	38.2 U/L

Appendix D. The arterial blood gas	
Measured Tests:	
pH @ 37 deg	7.447
PCO2 @ 37 deg	37.7 mmHg
PO2 @ 37 deg	172.4 mmHg
TempM	27.0 deg
Calculated Tests:	
BE-ecf	2.0 mmol/L
BE-b	2.9 mmol/L
SBC	27.0 mmol/L
HCO3-	26.3 mmol/L
A	103.5 mmHg
a/A	1.7
PO2/FIO2	824.9 mmHg
TCO2	27.4 mmol/L
SO2%	99.6 %

Appendix E. The 12-lead electrocardiogram	
Rate: 55 beats per minute	PR interval: 0.20 seconds
Rhythm: Sinus	QRS: 0.08 seconds
Axis: 82 deg	ST segment: Isoelectric
P wave: biphasic with terminal negative portion > 0.04 seconds	QT: 0.40 seconds
	QTc: 0.39 seconds
Impression: Sinus bradycardia, left ventricular hypertrophy by Cornell criteria, left atrial abnormalities	

Appendix F. The chest radiography taken postero-antero-lateral view

Findings:
- The heart is enlarged with inferolateral displacement of the cardiac apex, prominence of the left atrial appendage, and left main pulmonary artery with loss of the cardiac waistline.
- Both hemidiaphragms are intact. Both costophrenic sulci are widened.
- The rest of the included structures are unremarkable.

Impression:
- Left-sided cardiomegaly with mild pulmonary congestion. 2D echo correlation suggested.

Appendix G. The preoperative transthoracic echocardiogram

Description:
- Normal left ventricular size and wall thickness with adequate wall motion and contractility. The left ventricular ejection fraction is 69%.
- Normal right ventricular size with good contractility.
- Dilated left atrium.
- Normal right atrial size.
- Thickened anterior mitral valve leaflet with diastolic doming motion. The posterior mitral valve leaflet is likewise thickened and is upright. Both commissures are partially fused.
- Structurally normal aortic valve and pulmonic valve.
- Normal main pulmonary artery.
- Normal aortic root dimension.
- No intracardiac thrombus.
- No pericardial effusion.

Doppler:
- There is abnormal mosaic color flow across the mitral valve and tricuspid valve.
- Estimated pulmonary artery systolic pressure of 76 mmHg by tricuspid regurgitation jet method.

Conclusion:
- Mitral stenosis, severe with mitral valve area of 0.65 cm^2 by planimetry and 0.83 cm^2 by pressure half time with peak gradient of 27 mmHg and mean gradient of 15 mmHg.
- Mitral regurgitation, severe.
- Tricuspid regurgitation, moderate.
- Normal left ventricular dimension with normal systolic function.
- Normal right ventricular dimension with good contractility.
- Dilated left atrium.
- Moderate pulmonary hypertension.

Appendix H. The postoperative transthoracic echocardiogram

Interpretation:
- Status post mitral valve replacement.
- Metallic prosthetic valve in mitral position with good opening and closing motion with effective orifice area of 2.3 cm^2 with gradient of 6.2 mmHg.
- Trivial mitral regurgitation.
- Trace aortic regurgitation.
- Severe tricuspid regurgitation.
- Normal left ventricular dimension with normal wall motion and contractility. Normal systolic function.
- Normal left atrium.
- Normal right atrium, aortic root, and main pulmonary artery dimensions.
- Structurally normal aortic valve, tricuspid valve, and pulmonic valve.
- Moderate pulmonary hypertension (66 mmHg) by tricuspid regurgitation jet.

When compared with previous study, the following changes are noted:
- There is now metallic prosthetic valve at the mitral valve position.
- Mitral regurgitation decreased from severe to trivial.
- Tricuspid regurgitation increased from moderate to severe.
- Left atrium decreased from 4.7cm^2 to 3.5cm^2.

Appendix I. The left heart catheterization and coronary angiogram report

Technique: The right femoral artery was accessed win the Modified Seldinger's technique. Coronary angiogram was done using 5F FL4.0 and 6F FR4.0 diagnostic catheters.
Fluoroscopy: Calcifications noted at the mitral valve area.
Left heart catheterization: LVEDP of 6 mmHg without gradient on pull back.
Coronaries: Type III LAD
- Left main coronary artery: 3.0 mm normal vessel
- Left anterior descending artery: 3.0 mm normal vessel
- Left circumflex: 2.75 mm normal vessel
- Right coronary artery: 3.0 mm normal vessel. The RPDA and PLB are good sized normal vessels

Impression:
- Angiographically normal coronary arteries.

Appendix J. The computed tomography scan with contrast of the neck

Findings:
- Multiple plain and IV contrast-enhanced axial CT images of the neck were obtained. No adverse reactions were observed.
- No definite mass lesions or abnormalities enhancing areas appreciated.
- The nasopharyngeal space is clear. Bilateral fossae of Rossenmuller are symmetric. Torus tobarius are likewise bilaterally intact.
- The oropharynx, oral cavity, laryngopharynx, and tracheal air column are normally patent with no abnormal luminal densities.
- Paranasal sinuses, nasal cavities, and turbinates are unremarkable. Nasal septum is at the midline.
- Visualized deep and superficial spaces of the suprahyoid and infrahyoid neck are clear.
- Epiglottis, pyriform sinuses, laryngeal cartilages, and vocal cords reveal no focal lesions.
- No cervical lymphadenopathy demonstrated. Visualized osseous structures are unremarkable.
- No other significant findings.

Impression:
- Negative neck study.

Appendix K. The surgical pathology report
Specimen: - Mitral valve.
Diagnosis: - Fibrosis and dystrophic calcification.