Case Report

ORTNER'S SYNDROME CASE REPORT: 53 YEARS OLD FEMALE PATIENT OF VALVULAR HEART DISEASE WITH HOARSENESS OF VOICE.

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INTRODUCTION:

Hoarseness of voice that occurs in cardiovascular disorders is known as ortner cardiovocal syndrome or simply ortner syndrome described first time by a Viennese physician Nobert Ortner in 1897 in patients with mitral stenosis.[1] There are many conditions that can lead to this syndrome including neoplastic , infectious, metabolic, traumatic or idiopathic. The cardiovascular disorders related to the syndrome are described in table 1. [2,3] Here we describe a case of 53 years old female known case of rheumatic heart disease presented with hoarseness of voice.

CASE REPORT

A 53 years old female patient was admitted in our hospital through outpatient department with history of progressive shortness of breath, palpitation and hoarseness of voice for last ten years. She was diagnosed as a case of rheumatic heart disease ten years ago when she started having shortness of breath on moderate exertion along with palpitations that worsened over time and now for last nine months she cannot do mild exertion without having shortness of breath and palpitations with episodes of orthopnoea and paroxysmal nocturnal dyspnoea. She started having hoarseness of voice ten years ago that was initially intermittent but for last six to seven months she had to speak forcefully to communicate. She was known hypertensive for last ten years on irregular antihypertensive therapy and asthmatic for which she was never worked up. She did not give any history of sore throat and high grade intermittent fever in her childhood. Her family history is not significant for ischemic heart disease. She is postmenpausal for last five years and mother of seven kids with no history of any event during and post delivery. She was on oral frusimide 40mg twice daily, aldactone 50 mg digoxin 0.25 mg , potassium chloride 500mg twice daily and nebulization with clenil and ventoline.

Table 1. Cardiovascular Conditions Associated With Ortner's / Cardiovocal Syndrome.

Congenital	Atrial or ventricular septal defect, double outlet right ventricle, Eisenmenger's complex, patent ductus arteriosus, Ebstein's anomaly, aortopulmonary window
Mitral valve disorders	Mitral stenosis, regurgitation, prolapsed
Aortic aneurysms	Saccular, atherosclerotic, pseudoaneurysms, dissections, traumatic, mycotic
Adult cardiovascular disorders	Left atrial enlargement, left ventricular aneurysm, pulmonary hypertension, ductus aneurysm, pulmonary embolism, thrombosed giant left atrium, tortuosity of great vessels, atrial myxoma
Iatrogenic	Cardiac or thoracic surgery, defibrillation, atrial fibrillation ablation procedure, repair of thoracic aneurysms
Miscellaneous	Foreign body causing oesophago-broncho-aortic fistula

On examination patient looked normal with mild respiratory distress with no peripheral oedema, clubbing or cyanosis. Her BP was 140/80, pulse rate was 99/min irregularly irregular, respiratory rate was 16/min and 98.6^{0}F temperature. The head and neck examination showed raised JVP. Cardiovascular examination revealed a tapping apex beat in left sixth intercostals space in midclavicular line. Left parasternal heave and a palpable second heart sound. On auscultation, the first heart sound was loud and the pulmonary component of the second heart sound was prominent at the apex and pulmonary area respectively. A grade 3/6 rough rumbling diastolic murmur was heard at the mitral area. Another grade 3/6 harsh ejection systolic murmur was audible in right upper parasternal space with radiation into ipsilateral carotid. The chest had bilateral rhonchi and oc-

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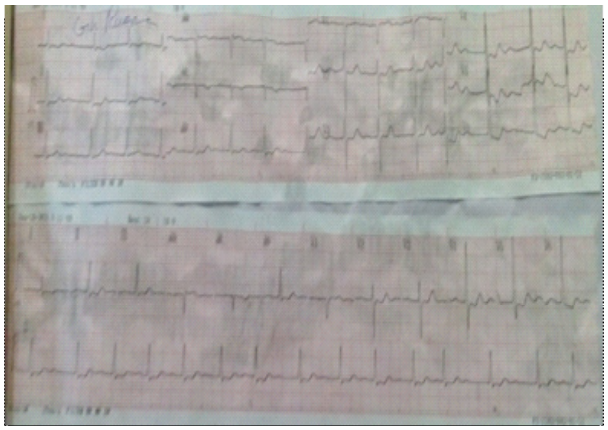
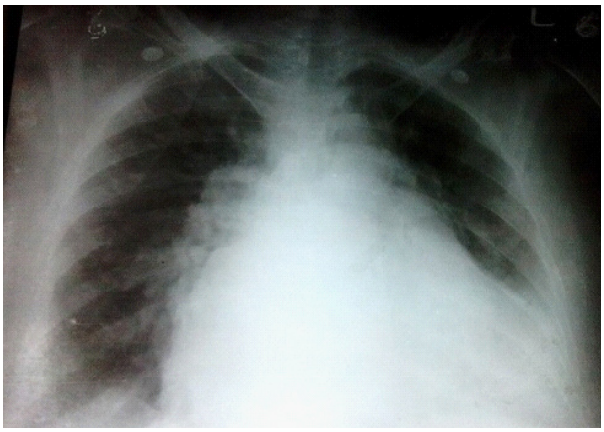


Figure 1



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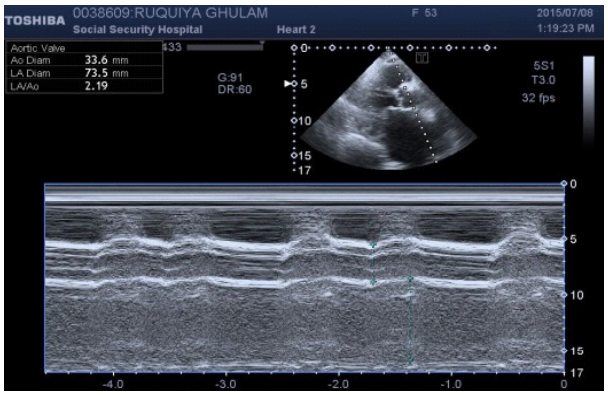

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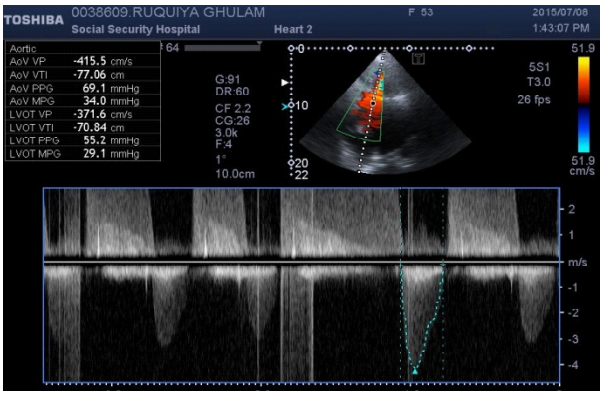

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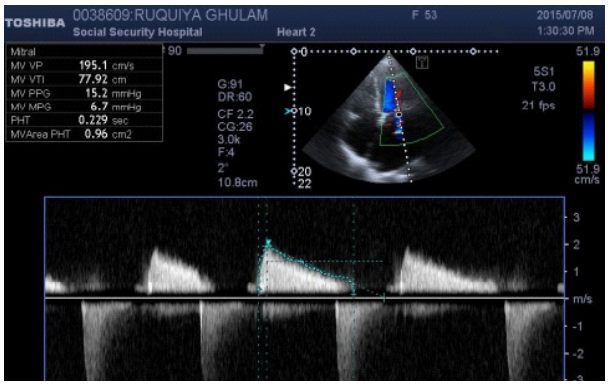

Figure 4

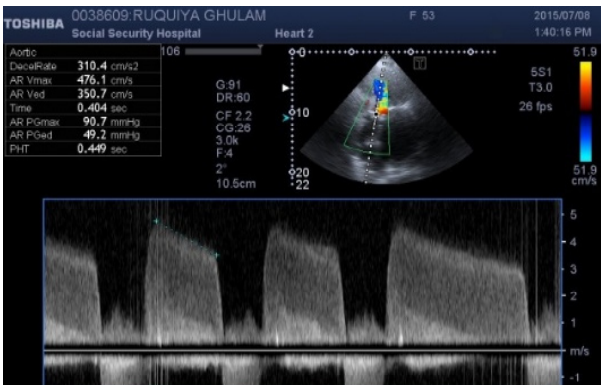

Figure 6

casional coarse crepts. Abdominal examination was normal with no visceromegaly.

Her hemoglobin was 12.0 g/l, total white cell count was 5.2 × 109/l and platelet count was 162 × 109/l. ESR was 46 mm in the first hour. Blood urea was 30 mg/dl, sodium was 135 mmol/l, potassium was 3.8 mmol/l and serum creatinine was 0.7 mg/dl. SGPT was 30 U/l, SGOT 29 U/l and INR of 2.5.

Electrocardiogram showed irregular rhythm at rate of 90/min with no visible p waves and signs of left ventricular hypertrophy with ST depression and asymmetrical T wave inversion in lateral leads with normal axis (fig 1). Chest radiograph demonstrated increased cardiothoracic ratio with left atrial enlargement and enlarged pulmonary arterial trunk and prominent pulmonary vascular markings and upper lobe diversion (fig 2). Transthoracic echocar-

diography showed thickened rheumatic mitral valve with fixed PML. The mitral valve area was 0.9 cm^2 using calculation from pressure half-time and mild mitral regurgitation. The left atrium was enlarged (left atrial diameter = 8.0 cm). There was no left atrial thrombus. The right ventricle was borderline enlarged with good function. Aortic valve was thickened calcified with restricted opening with moderate aortic stenosis having mean gradient of 37mmHg and mild to moderate AR with ARPHT 523 ms (fig 3-6). Moderately severe pulmonary hypertension with TVPG of 50mmHg. Both right and left ventricular function were normal with mild concentric LVH. Direct laryngoscopy performed by the ENT surgeon revealed a left vocal cord paralysis that was fixed in paramedian position. Coronary angiography revealed normal coronaries. She was advised surgery with dual valve replacement.

DISCUSSION:

Recurrent laryngeal nerve is a branch of vagus nerve that loops around under the aortic arch on left side and subclavian artery on right side and ascends back to the neck in the tracheoesophageal groove to supply the muscles of larynx except cricothyroid(cricothyroid supplied by superior laryngeal nerve).[4] Left recurrent laryngeal nerve is usually involved in cardiovascular disorders and its palsy leads to left vocal cord to be fixed in paramedian position causing loss of air and shortness of breath while speaking , dysphonia and hoarseness of voice. It was initially thought that enlarged left atrium causes compression of left recurrent laryngeal nerve under the arch of aorta but subsequent studies and autopsy data showed that compression of left recurrent laryngeal nerve between arch of aorta and pulmonary artery is responsible for this palsy.[4, 5]

Ortner syndrome is a rare complication of rheumatic heart disease because of early intervention and is thought to be caused by giant left atrium (GLA). Giant left atrium is defined as left atrial diameter more than 65 mm on echocardiography.[6] Left atrial enlargement can cause compression of left bronchus, left ventricle, oesophagus, middle and inferior lobes of right lung, and left recurrent laryngeal nerve leading to dyspnoea, heamodynamic instability, dysphagia, atelactesis, and hoarseness of voice respectively.[7] Hoarseness of voice in this patient is secondary to left pulmonary artery being pushed up against aortic arch by enlarged left atrium leading to compression of left recurrent laryngeal nerve between two vessels. The treatment strategy in such patients is mitral valve surgery with or without left atrial volume reduction to undo the effects of compression according to surgeon's experience.[8]

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