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Effect of Ayurvedic Add on Management in Adulthood Bronchial Asthama Leading to Risk of Coronary Artery Disease in Old Age - A Single Case Study

Dr. Fareeda Begum Shaikh[1], Dr. Sumayya Begum[2]

[1]Professor, Department of Kayachikitsa, TGAMC, Ballari, Karnataka, India
[2]PG Scholar, Department of Kayachikitsa, TGAMC, Ballari, Karnataka, India

ABSTRACT

Introduction: Coronary Artery Disease involves atherosclerotic plaque formation in lumen causing impairment of blood flow to myocardium and is a major cause of death having incidence of 7%–13% urban and 2–7% rural. Persistent pulmonary inflammation leads to low-grade systemic inflammation, influencing blood vessels, triggering coronary artery disease (CAD) and release of pro-inflammatory mediators and cytokines into the circulation stimulate the production and release of acute-phase proteins and inflammatory cells, resulting in a state of low-grade systemic inflammation and this has an impact on blood vessels, encouraging the instability and rupture of atherosclerotic plaque and precipitating sudden cardiac and cerebral events. There is even significant influence of anti-asthmatic medications on CAD development.

Materials and Methods: An open labelled, single-arm, prospective observational case study. A 74-year-old male complained of hrudshoola, arohana ayasa, padashotha, parshvashoola and kasa since 3 years with a history of adulthood asthma and hypertension (15 years) and was on medication for the same. He was diagnosed with CAD – triple vessel disease s/p CABG, mild LV systolic dysfunction and grade I diastolic dysfunction. He visited Kayachikitsa OPD TGAMC, Ballari. On the same day, spirometry showed severe bronchial obstruction with normal chest X-ray. He was treated with Tulasi churna 1 tsf OD with ushna jala before food for 7 days. After attaining nirama avastha, sadyovirechana with Nimbamrutadi castor oil 50 ml with 100 ml milk was given and number of vegas were 8. Next day, sthanika abhyanga over urah pradesha with salavana murchita tila taila followed by nadisweda was done for 7 days. Then shamanaushadhi like Hinguvachadi churna 1 tsf TID with ushna jala before food, Punarnavasava 60 ml TID with 60 ml ushna jala after food and Prabhakara vati 2 BD with ushna jala after food as hrudrasayana was given for 69 days.

Observations and Results: Subjective improvement was seen in shotha, ayasa and hrudshoola, and objective improvement was seen in spirometry with changes from severe obstruction to moderate obstruction, with improvement in FEV1/FVC from 56% to 72%.

Discussion and Conclusion: Alkaloids and glycosides present in Tulasi churna act as cardiostimulants. It is a proven bronchodilator and immunomodulator. Tulasi, due to its teekshna and ushna guna, acts as vata-kapha hara and shwasa hara, and due to tikta guna acts as hridya. Salavana taila sthanika abhyanga and nadi sweda cause kaphavilayana and their swedana effect causes vasodilation, thereby improving blood flow and correcting oxygen mismatch. Hinguvachadi churna acts as vaatanulomana, shothahara, hridshoolahara and does srotovishodhana, thus correcting vimarga gamana of udana and vyana vata. Punarnavasava is anti-inflammatory, shothahara, acts on high cholesterol and is kasahara. Prabhakara vati is a hrudrogahara rasayana, does hrudbalya and due to its yogavahi property acts on high cholesterol, asthma and inflammation.

KEYWORDS: *CAD, Asthma, Tulasi churna, Salavana taila, Nadisweda, Nimbamrutadi castor oil, Hinguvachadi churna, Punarnavasava, Prabhakara vati.*

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INTRODUCTION

Coronary artery disease involves atherosclerotic plaque formation in the lumen of vessel causing impairment of oxygen supply to myocardium resulting in demand supply mismatch of oxygen. It is multifactorial phenomenon involving modifiable and nonmodifiable etiological factors. Modifiable risk include smoking, obesity, lipid level, psychosocial and asthma, COPD. Nonmodifiable include gender, age, family history and genetics. CAD represented 2.2% of overall global burden of disease and 32.7% of cardiovascular diseases.

Asthma is prevalent chronic inflammatory airway disease and an increased risk of coronary artery disease. Recent systemic reviews suggest that the risk of coronary artery disease and heart failure are two fold higher in individual with asthma.

Association of asthma and CAD is inconsistent and poorly understood. Mechanism that link asthma and CAD are Systemic inflammation, Persistent inflammation present in asthma contributes to formation of atherosclerosis. Oxidative stress damages vascular endothelium. Repeated episodes of airway obstruction may lead to cardiovascular stress and long term use of corticosteroids, beta agonists and other medication impacts cardiovascular health. There is emerging challenge as increased prevalence of CAD are noticed and cost effective and efficient treatments are lagging in developing countries. Need for the study is to understand pathophysiology of plaque formation and persistent inflammation and immune response involved in CAD development in chronic asthmatic patient, Hence a single prospective observational case study done in adulthood asthmatic patient who developed CAD in old age with Ayurvedic intervention - A classical treatment modality.

Materials and method

Study Design: Prospective Open Labelled single arm Case Study.

Chief complains

A 74 years old male complaints of hrudshoola, arohana ayasa, padashotha, parshvashoola and kasa since 3 years with the history of adulthood asthama and Hypertension (15 years) was on medication for the same, diagnosed with CAD-TRIPPLE VESSEL DISEASE S/P CABG, Mild LV Systolic dysfunction and Grade I Diastolic dysfunction has visited Kayachikitsa OPD TGAMC Ballari and on the same day Spirometry showed severe bronchial obstruction with normal chest X-ray.

Personal History

Ahara: Katu Ushna Ruksha
Vyasana: Divaswapna
Tea 2 times/day
Nidra: Disturbed
Mala: gratitha
Mutra: 6/7times /day
Krodha shoka atichintana

General examination

Height: 5'7"
Weight: 65
BMI: 21
Built: moderate
Nutrition: moderate
Pallor, Edema: present

Systemic Examination

Respiratory system
Wheezing B/L present ++

CVS System
S1 S2 heard
BP: 160/120
PR: 68bpm

Roga Parīkṣā

Śvāsa is a Prāṇavaha srotoduṣṭi-janya vyādhi, predominantly involving Vāta and Kapha doṣas. When Śvāsa becomes chronic, recurrent, or improperly managed, it gradually affects the Hṛdaya, the mūlasthāna of Prāṇavaha srotas, ultimately resulting in Hṛdroga.

The repeated provocation of Prāṇa vāyu by Kapha, āma, and srotorodha in the ūrdhva śarīra causes forceful and labored respiration. Chronic Prāṇa–Udāna vāyu vaishamya leads to persistent stress on the Hṛdaya, which is the seat of Prāṇa, Ojas, Manas, and Rasavaha srotas.

Long-standing Śvāsa causes:

- Rasa dhātu duṣṭi due to impaired oxygenation and circulation
- Ojakṣaya due to continuous respiratory distress
- Hṛdaya gaurava and hṛd-śūla due to Kapha–Vāta āvaraṇa

As Kapha accumulates in the uras and hṛdaya pradeśa, it obstructs the normal gati of Prāṇa vāyu. This Prāṇavaha srotorodha results in hypofunctioning of Hṛdaya, leading to symptoms such as hṛdgraha, hṛdspandana vaishamya, śvāsa-kṛcchrata, and ālasya, thereby manifesting as Hṛdroga secondary to Śvāsa.

With chronicity, Vāta prakopa dominates, causing hṛdaya kṣobha, rūkṣatā, and dhātukṣaya, culminating in Vātaja or Sannipātaja Hṛdroga. Thus, untreated or long-standing Śvāsa acts as a nidāna for Hṛdroga, especially in individuals with kṣīṇa bala, durbala ojas, and pre-existing srotovaiguṇya.

Samprapti Ghāṭaka

Doṣa	Prāṇa Vāta (pradhāna), Udāna Vāta; Kapha (āvaraka); later Vāta-pradhāna Sannipāta
Dūṣya	Rasa, Rakta, Ojas
Agni	Jatharāgni mandya; Rasa dhātvagni mandya
Āma	Present, especially in early and Kapha-dominant stages
Srotas	Prāṇavaha (mukhya), Rasavaha, Manovaha
Srotodusṭi Prakāra	Saṅga, Āvaraṇa, Vimarga gamana
Udbhava Sthāna	Āmāśaya, Pakvāśaya
Saṅcāra Sthāna	Uras, Prāṇavaha srotas
Adhiṣṭhāna	Hṛdaya
Vyakti Sthāna	Hṛdaya and Uras
Roga Mārga	Madhyama roga mārga
Roga Svabhāva	Cira-kārī, Yapya to Kaṣṭha-sādhya
Prabhāva on Ojas	Ojakṣaya due to chronic Śvāsa and Prāṇa vāyu kṣobha

Investigation

1. 2D echo:

S/P CABG
Mild LV systolic dysfunction
Grade 1 LV Diastolic dysfunction
Trivial MR Mild AR
No PE/ clot/ vegetation

2. CHEST XRAY

Soft tissues and thoracic cage are normal.
Cardiothoracic Ratio within normal limits.
Both hila are normal.

3. Spirometry

FEV1/FVC 56% severe obstruction

Present ongoing modern Medication

1. Tab. Paracetamol 650mg 6am-12pm-6pm-10pm
2. Tab. Clopitab-A 75mg once daily 2pm
3. Tab. Cardace 2.5mg once daily 8pm
4. Tab. Lasilactone 12.5mg once daily 8am
5. Tab. Metoprolol 12.5mg once daily Sam
6. Tab. Aztor 20mg once daily 8pm
7. Tab. Rantac 150mg twice daily 7am-7pm
8. Cap. Haemup once daily 2pm

Intervention.

S. No.	Therapeutic Measure	Drug / Procedure	Dose & Anupana	Time of Administration	Duration	Purpose
1	Deepana–Pachana	Tulasi Churna	1 teaspoon with Ushna Jala	Before food	7 days	To correct Agnimandya and digest Ama
2	Sadyovirechana	Nimbamrutadi Castor Oil	50 ml with 100 ml Ushna Jala	Before food	Single sitting	To eliminate Pitta–Kapha and clear Srotorodha
3	Sthanika Chikitsa	Abhyanga over Urah Pradesha with Salavana Murchita Tila Taila followed by Nadi Sweda	External application	Once daily	7 days	To pacify Vata, improve local circulation, and relieve Hridgata Vata

4	Shamana & Hridrasayana Chikitsa	Hinguvachadi Churna	1 teaspoon TID with Ushna Jala	Before food	69 days	Deepana, Vatanulomana
		Punarnavasava	60 ml with 60 ml Ushna Jala	After food	69 days	Hridya, Shothahara, Rasayana
		Prabhakara Vati	2 tablets BD with Ushna Jala	After food	69 days	Hridrasayana, Balya, Vata-Pitta shamana

Observation and results

Subjective parameters	Before treatment	After treatment
Hridshoola	+++	-
Arohana ayasa	+++	+
Swasakrichrata	+++	-
Shotha	++++	+

Objective parameters	Before treatment	After treatment
Blood pressure	160/120mmhg	130/100mmhg
Echo	S/P CABG Mild LV systolic dysfunction Grade 1 LV Diastolic dysfunction Trivial MR Mild AR No PE/ clot/ vegetation	
Spirometry	severe obstruction of FEV1/FVC 56%	Normal FEV1/FVC 77%

Discussion

Shwasa (adult-onset asthma) and Hrudroga (coronary artery disease) share a common pathological origin, as both arise from Hrudaya, which serves as the moolasthana of Pranavaha and Rasavaha srotas. Any disturbance at this level simultaneously affects respiration, circulation, and tissue nourishment. In the present case, the disease process was characterized by vimargagamana of Udana and Vyana Vata along with sanga of Kapha, leading to obstruction of airflow, impaired myocardial perfusion, and chronic inflammation. The involvement of Rasa and Rakta dhatus further contributed to endothelial dysfunction, dyslipidemia, and compromised oxygen delivery, creating a sustained cardio-pulmonary pathological continuum.

The initial therapeutic focus was directed toward correction of Agni and elimination of Ama, as impaired digestion and metabolism form the foundation for both Shwasa and Hrudroga. Deepana–Pachana with Tulasi churna was therefore administered.

Tulasi, by virtue of its katu and ushna qualities, facilitates Kapha vilayana and restores the normal gati of Vata, thereby relieving srotorodha. Its tikta rasa supports Hrudaya by enhancing functional strength and vitality. Phytochemical constituents such as eugenol, ursolic acid, rosmarinic acid, alkaloids, and glycosides contribute to bronchodilation, cardiostimulation, antioxidant activity, and immunomodulation. These actions collectively reduce airway hyperresponsiveness, improve myocardial contractility, and attenuate low-grade systemic inflammation, establishing a stable platform for further interventions.

Considering the persistence of Kapha-Pitta involvement and Vata avarodha at the level of koshta, Sadyovirechana was planned using Nimbamrutadi castor oil. This intervention enabled rapid koshtashodhana and vatanulomana, thereby interrupting the disease process at its root. The formulation, containing Nimba, Amruta, and Eranda, acts synergistically to reduce inflammation, correct metabolic imbalance, and eliminate accumulated morbid doshas. Eranda, known for its potent Vata-pacifying action, facilitates unobstructed movement of Vata, while bioactive compounds such as ricinoleic acid and diterpenoids exert anti-inflammatory, lipid-lowering, and immunoregulatory effects. Through this mechanism, Sadyovirechana directly contributed to sampraptivighatana in both respiratory and cardiovascular pathology.

Local therapeutic measures were employed to address the site of pathology more directly. Abhyanga over the urah pradesha with Salavana murchita tila taila followed by Nadi Sweda produced combined snehana and swedana effects. This process led to the liquefaction and mobilization of grathita shleshma

adherent to the srotas, facilitating clearance of obstruction. As the channels became patent, the previously obstructed Vata, particularly Udana and Vyana, regained its physiological movement. Improved chest wall compliance, enhanced bronchial clearance, and better regional circulation were observed as a result of improved microcirculation, vasodilation, and lymphatic drainage induced by these therapies.

For sustained internal management, Prabhakara Vati was administered as a Hrudrasayana. The formulation contains Makshika, Loha Bhasma, Abhraka Bhasma, and Shilajatu, which collectively strengthen cardiac musculature, enhance oxygen utilization, and improve Rakta dhatu quality. Shilajatu, rich in fulvic acid and dibenzo-α-pyrones, improves mitochondrial efficiency, reduces oxidative stress, and modulates lipid metabolism. These effects are particularly relevant in conditions marked by dyslipidemia, chronic inflammation, and compromised myocardial energy dynamics, thereby supporting its role in coronary artery disease and chronic respiratory disorders.

Punarnavasava was incorporated to address inflammation, edema, and metabolic disturbances aggravated by prolonged steroid exposure. Punarnava, along with Bruhati, Kantakari, and Vasaka, acts to reduce Shwasa and Kasa while simultaneously correcting fluid imbalance and lipid abnormalities. Active constituents such as punarnavine, flavonoids, and alkaloids exert anti-inflammatory, diuretic, bronchodilatory, and cardioprotective effects. This dual action on the respiratory and cardiovascular systems helped mitigate airway inflammation, reduce vascular edema, and improve functional capacity.

Hinguvachadi churna played a crucial role in maintaining Vatanulomana and preventing recurrence of obstruction. By correcting the abnormal directional movement of Udana and Vyana Vata, it ensured sustained patency of the srotas. Its shothahara and srotovishodhana actions reduced inflammatory changes within the bronchi and blood vessels, alleviating Hridshoola and respiratory distress. The presence of bioactive compounds such as ferulic acid and sulfur-containing resins contributed to antispasmodic, bronchodilatory, and vasodilatory effects, further stabilizing cardio-pulmonary function.

Overall, the therapeutic approach adopted in this case addressed the disease process at multiple levels, including metabolic correction, elimination of morbid factors, restoration of physiological Vata movement, clearance of obstructed channels, and long-term tissue rejuvenation. This comprehensive intervention not only provided symptomatic relief but also targeted the underlying pathophysiological continuum linking Shwasa and Hrudroga, resulting in sustained clinical improvement.

Conclusion:

CAD is a major cause of death in patients having comorbidities like high cholesterol, DM, COPD, and asthma. Both Shwasa and Hrudroga affect Hrudaya, and treatment targeting the health of the heart becomes essential. Hence, classical treatment of Salavana abhyanga followed by Nadisweda and Hrudrasayana like Hinguvachadi churna, Prabhakara vati, and Punarnavasava are given as add-on therapy to ongoing modern treatment.

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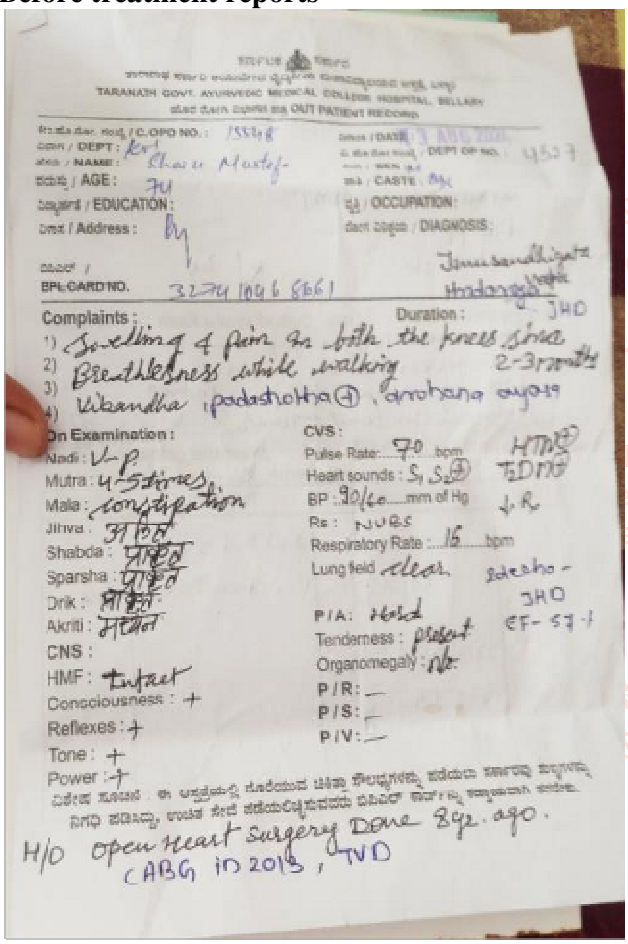
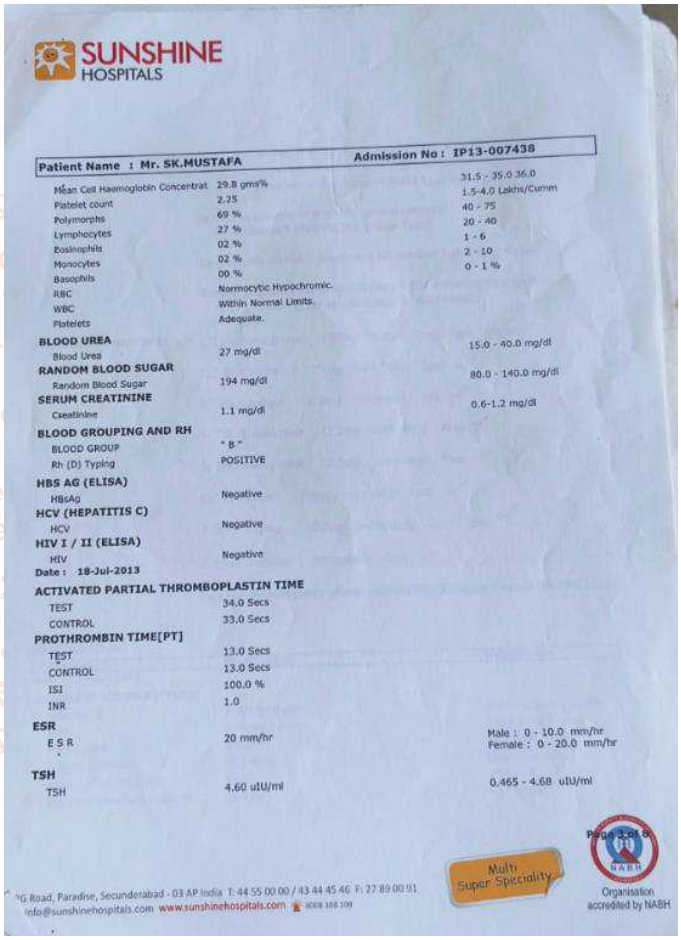
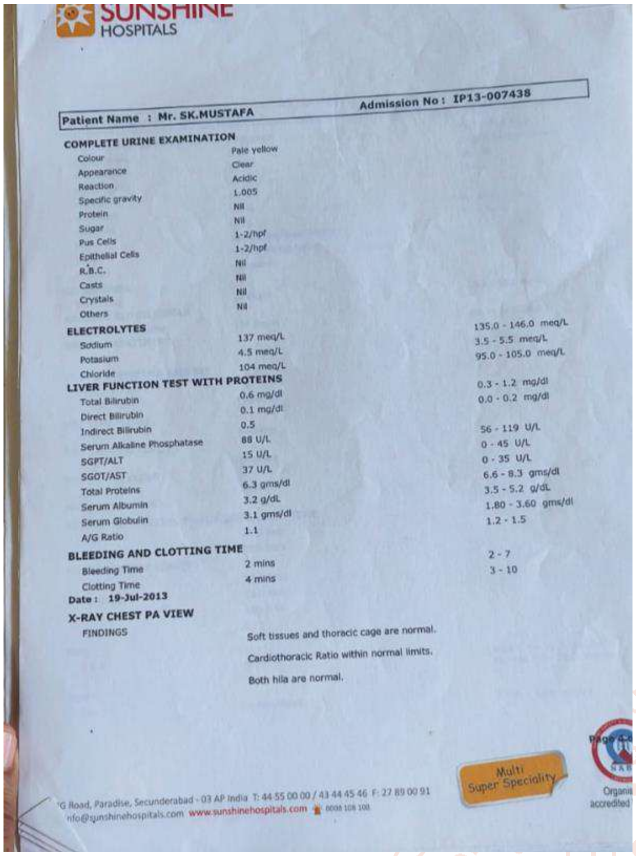
Before treatment reports







SUNSHINE HOSPITALS

Patient Name : Mr. SK.MUSTAFA Admission No : IP13-007438

COMPLETE URINE EXAMINATION

Colour	Pale yellow	
Appearance	Clear	
Reaction	Acidic	
Specific gravity	1.005	
Protein	Nil	
Sugar	Nil	
Pus Cells	1-2/hpf	
Epithelial Cells	1-2/hpf	
R.B.C.	Nil	
Casts	Nil	
Crystals	Nil	
Others	Nil	
ELECTROLYTES		
Sodium	137 meq/L	135.0 - 146.0 meq/L
Potasium	4.5 meq/L	3.5 - 5.5 meq/L
Chloride	104 meq/L	95.0 - 105.0 meq/L
LIVER FUNCTION TEST WITH PROTEINS		
Total Bilirubin	0.6 mg/dl	0.3 - 1.2 mg/dl
Direct Bilirubin	0.1 mg/dl	0.0 - 0.2 mg/dl
Indirect Bilirubin	0.5	
Serum Alkaline Phosphatase	88 U/L	56 - 119 U/L
SGPT/ALT	15 U/L	0 - 45 U/L
SGOT/AST	37 U/L	0 - 35 U/L
Total Proteins	6.3 gms/dl	6.6 - 8.3 gms/dl
Serum Albumin	3.2 g/dL	3.5 - 5.2 g/dL
Serum Globulin	3.1 gms/dl	1.80 - 3.60 gms/dl
A/G Ratio	1.1	1.2 - 1.5
BLEEDING AND CLOTTING TIME		
Bleeding Time	2 mins	2 - 7
Clotting Time	4 mins	3 - 10

Date : 19-Jul-2013

X-RAY CHEST PA VIEW

FINDINGS

Soft tissues and thoracic cage are normal.

Cardiothoracic Ratio within normal limits.

Both hila are normal.

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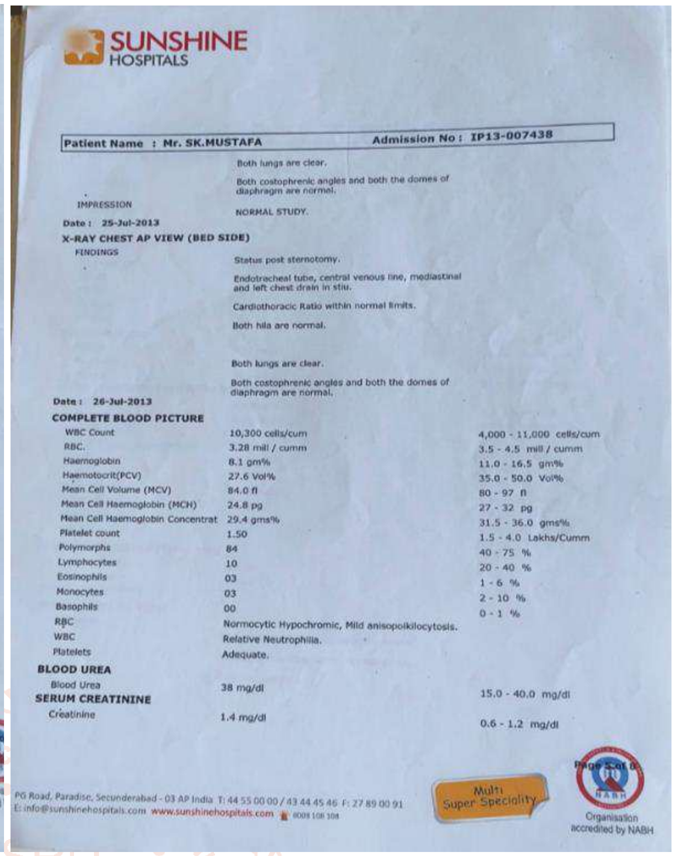

SUNSHINE HOSPITALS

Patient Name : Mr. SK.MUSTAFA Admission No : IP13-007438

Both lungs are clear.

Both costophrenic angles and both the domes of diaphragm are normal.

IMPRESSION NORMAL STUDY.

Date : 25-Jul-2013

X-RAY CHEST AP VIEW (BED SIDE)

FINDINGS

Status post sternotomy.

Endotracheal tube, central venous line, mediastinal and left chest drain in situ.

Cardiothoracic Ratio within normal limits.

Both hila are normal.

Both lungs are clear.

Both costophrenic angles and both the domes of diaphragm are normal.

Date : 26-Jul-2013

COMPLETE BLOOD PICTURE

WBC Count	10,300 cells/cum	4,000 - 11,000 cells/cum
RBC.	3.28 mill / cumm	3.5 - 4.5 mill / cumm
Haemoglobin	8.1 gm%	11.0 - 16.5 gm%
Haemotocrit(PCV)	27.6 Vol%	35.0 - 50.0 Vol%
Mean Cell Volume (MCV)	84.0 fl	80 - 97 fl
Mean Cell Haemoglobin (MCH)	24.8 pg	27 - 32 pg
Mean Cell Haemoglobin Concentrat	29.4 gms%	31.5 - 36.0 gms%
Platelet count	1.50	1.5 - 4.0 Lakhs/Cumm
Polymorphs	84	40 - 75 %
Lymphocytes	10	20 - 40 %
Eosinophils	03	1 - 6 %
Monocytes	03	2 - 10 %
Basophils	00	0 - 1 %
RBC	Normocytic Hypochromic, Mild anisopoikilocytosis.	
WBC	Relative Neutrophilia.	
Platelets	Adequate.	
BLOOD UREA		
Blood Urea	38 mg/dl	15.0 - 40.0 mg/dl
SERUM CREATININE		
Creatinine	1.4 mg/dl	0.6 - 1.2 mg/dl

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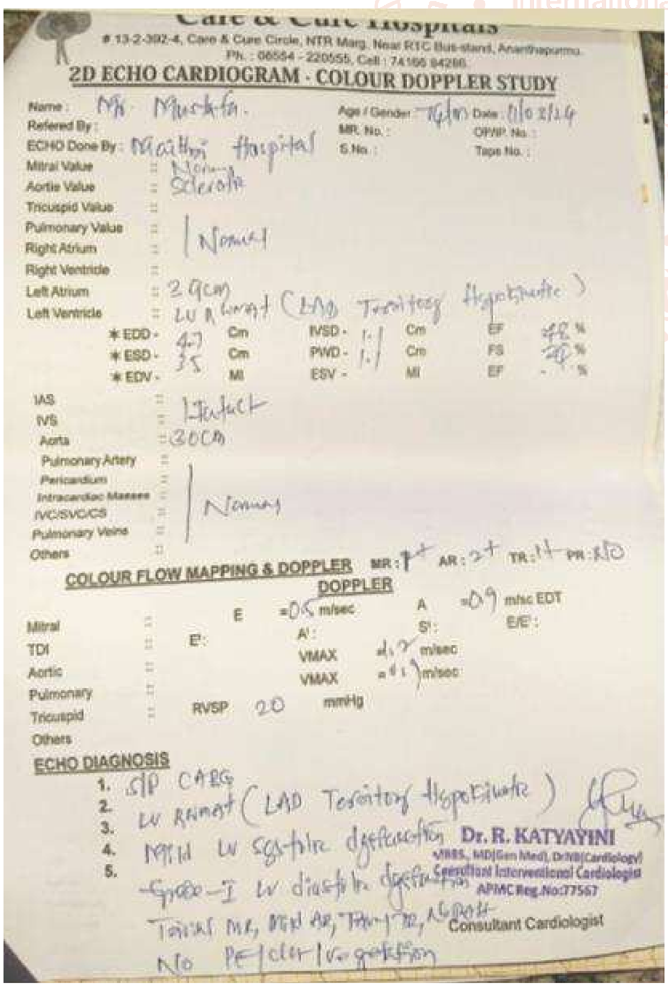
After treatment reports



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13-2-392-4, Care & Cure Circle, NTR Marg, Near RTC Bus-stand, Ananthapuramu.

2D ECHO CARDIOGRAM - COLOUR DOPPLER STUDY

Name : Mr. Mustafa. Age / Gender : Date :
Refered By : MR. No. : OP/IP. No. :
ECHO Done By : Maithri Hospital S.No. : Tape No. :

Mitral Value : Normal
Aortic Value : Sclerotic
Tricuspid Value :
Pulmonary Value : Normal
Right Atrium :
Right Ventricle :
Left Atrium : 2.9cm
Left Ventricle : LV Rwmat (LAD Territory Hypokinetic)

*EDD - 4.7 Cm IVSD - 1.1 Cm EF %
*ESD - Cm PWD - 1.1 Cm FS %
*EDV - 3.5 Ml ESV - Ml EF - %

IAS : Intact
IVS :
Aorta : 3.0cm
Pulmonary Artery :
Pericardium :
Intracardiac Masses : Normal
IVC/SVC/CS :
Pulmonary Veins :
Others :

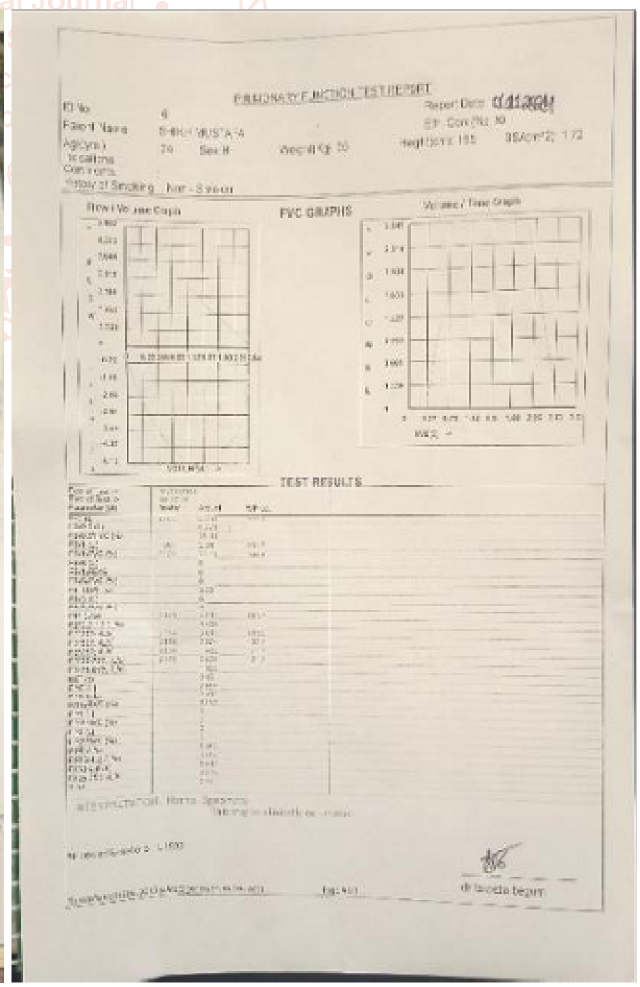
COLOUR FLOW MAPPING & DOPPLER MR : 1+ AR : 2+ TR : 1+ PR : NO

DOPPLER

Mitral : E =0.5 m/sec A =0.9 m/sec EDT
TDI : E' : A' : S' : E/E' :
Aortic : VMAX m/sec
Pulmonary : VMAX m/sec
Tricuspid : RVSP 20 mmHg
Others

ECHO DIAGNOSIS
1. S/P CABG
2. LV RWMAT (LAD Territory Hypokinetic)
3. Mild LV systolic dysfunction
4. Grade-I LV diastolic dysfunction
5. Trivial MR, Mild AR, Trivial TR, AS
No PE/clot/vegetation

Dr. R. KATYAYINI
Consultant Interventional Cardiologist
Consultant Cardiologist



PULMONARY FUNCTION TEST REPORT

FVC GRAPHS

Volume / Time Graph

TEST RESULTS

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