DISSERTATION

ON

A STUDY ON CARDIOVASCULAR MANIFESTATIONS

IN PATIENTS WITH

SYSTEMIC LUPUS ERYTHEMATOSUS

M.D. DEGREE EXAMINATION

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CERTIFICATE

This is to certify that dissertation entitled "**A STUDY ON CARDIOVASCULAR MANIFESTATIONS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS** **"** is the bonafide record of work done by **Dr. R.VIJAI ANANTH** in the Department of General Medicine , Thanjavur Medical College , Thanjavur during his Post Graduate Course from 2008 – 2011 . This is submitted as partial fulfilment for the requirement of M.D. Degree Examinations – Branch I (General Medicine) to be held in APRIL 2011.

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DECLARATION

I, **Dr. R.VIJAI ANANTH** solemnly declare that dissertation titled "**A STUDY ON CARDIOVASCULAR MANIFESTATIONS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS "** is a bonafide work done by me at Thanjavur Medical College, Hospital during JUNE 2009 – OCTOBER 2010 under the guidance and supervision of **Prof. DR.S.MUTHUKUMARAN.M.D.,** Prof and HOD, Department of Internal Medicine. The dissertation is submitted to **THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY, CHENNAI TAMILNADU** as partial fulfilment for the requirement of M.D. Degree Examinations – Branch I (General Medicine) to be held in April 2011.

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INTRODUCTION

Systemic Lupus Erythematosus (SLE) is an autoimmune disease in which organs and cells undergo damage mediated by tissue-binding auto antibodies and immune complexes.[1]

INCIDENCE AND PREVALENCE

The overall prevalence of SLE varies from 12 to 50.8 cases per 1 lakh persons.[4]

Three British groups who used several sources to ascertain cases arrived at prevalence rates ranging from 24.7 to 26.1 per 1 lakh persons[5].

The average annual incidence of SLE in united states vary from 2.0 to 7.6 cases per 1 lakh persons per year.[4] Prevalence of SLE in the united states is 15 – 50 per 1 lakh persons.[1]

In a study conducted near Delhi, the prevalence of SLE was found to be 3.2 per 1 lakh population.[3]

AGE AND SEX DISTRIBUTION

Ninety percent of patients are women of child-bearing age. people of both sexes, all ages, and all ethnic groups are susceptible.[1]

Female to male ratio is 9:1 between menarche and menopause, 3:1 in young and old .[2]

Age specific incidence rates in black and Caucasian females were greatest in 15 – 44 year age group.[4]

PATHOGENESIS AND ETIOLOGY

SLE is a multigenic disease[1]. Interactions between susceptibility genes and environmental factors result in abnormal immune responses. Those responses include

1. Activation of innate immunity .
2. Lowered activation thresholds of adaptive immunity cells .
3. Ineffective regulatory and inhibitory CD4+ and CD8+ T cells.
4. Reduced clearance of apoptotic cells and of immune complexes.[1]

Self antigens are available for recognition by the immune system in the surface blebs of apoptotic cells; thus antigens, antibodies and immune complexes persists for prolonged period of time , allowing inflammation and disease to develop.[1]

CLINICAL MANIFESTATIONS OF SLE AND PREVALENCE.[1]

Manifestation	Prevalence(%)
Musculoskeletal	95%
Cutaneous	80%
Hematologic	85%
Neurologic	60%
Cardiopulmonary	60%
Renal	30-50 %
Gastrointestinal	40%
Thrombosis	15%
Ocular	15%

AUTOANTIBODIES IN SLE.[1]

Antinuclear antibodies:

Anti-dsDNA

Anti-sm

Anti-RNP

Anti-Ro

Anti-La

Antihistone

Antiphospholipid

Antierythrocyte

Antiplatelet

Antineuronal

Antiribosomal P

1997 UPDATE OF THE 1982 AMERICAN COLLEGE OF RHEUMATOLOGY CLASSIFICATION CRITERIA FOR SLE.[6]

1. Malar rash

Fixed erythema, flat or raised, over the malar eminences.

2. Discoid rash

Erythematous circular raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur.

3. Photosensitivity

Skin rash as a result of unusual reaction to sun light, by patient history or physician observation.

4. Oral ulcers

Oral or nasopharyngeal ulceration, usually painless, observed by a physician.

5. Arthritis

Non erosive arthritis involving two or more peripheral joints, characterized by tenderness swelling or effusion.

6. Serositis

Pleuritis**:** convincing history of pleuritic pain or rub or evidence of pleural effusion.

or

Pericarditis: documented by ECG or rub or evidence of pericardial effusion.

7. Renal Disorder

Persistant proteinuria > 0.5 g / day or more than or equal to 3+ if quantitation not performed.

or

Cellular casts: may be red cell, haemoglobin, granular, tubular, or mixed.

8. Neurologic disorder

Seizures or Psychosis: in the absence of offending drugs or known metabolic derangement.

9. Hematologic Disorder

Haemolytic anaemia: with reticulocytosis or

Leukopenia : less than 4000/ mm^3 or

Lymphopenia less than 1500/mm^3 or

Thrombocytopenia less than 100,000/mm^3 in the absence of offending drug.

10. Immunologic disorder

Anti – DNA: Antibody to native DNA in abnormal titer.

or

Anti – sm: presence of antibody to sm nuclear antigen.

or

Positive finding of antiphospholipid antibodies.

11. Positive antinuclear antibody.

An abnormal titer of ANA by immunofluorescence or an equivalent assay at any point in time and in the absence of drugs known to be associated with drug induced lupus syndrome.

If more than or equal to 4 of these criteria, well documented, are present at any time in a patient's history the diagnosis is likely to be SLE. Specificity is 95%; sensitivity is 75%.[1]

AIMS OF THE STUDY

1. To find out the prevalence of cardiac manifestations in patients with Systemic Lupus Erythematosus.

2. To find out the commonest and least common cardiac manifestation in Systemic Lupus Erythematosus.

3. To find out the various types of cardiac manifestations in Systemic Lupus Erythematosus.

4. To compare the results of this study with the results of other studies in the literature.

REVIEW OF LITERATURE

The words of Brigden et al written in 1960, remain true today: "Heart lesions develop in nearly all SLE patients at some time during the course of their disease when life is prolonged by modern therapy.[7]

HISTORY

The pathologic study of cardiac lupus dates from the report of Libman and sacks of verrucous endocarditis.[8]

The first recognition of cardiac involvement in lupus was a report by kaposi in 1872 of cardiac irregularity and dyspnea.[9]

CARDIAC MANIFESTATIONS OF SLE

1. PERICARDITIS

Pericarditis tends to be one of the earlier cardiac manifestations and can even be the first manifestation of lupus.[10]

Pericarditis is the most frequent cardiac manifestation.[1]

Pericarditis usually appears as an isolated attack or as recurrent episodes, with or without symptoms.[7]

In a French series, of 28 patients with pericarditis, 23 had pain, 12 had rub, and 4 required pericardiocentesis because of tamponade.[11]

Patients with pericardial effusion are more likely to have pericardial pain and active lupus.[12] Pericardial tamponade has been reported.[10] constrictive pericarditis is very rare.[13]

PREVALENCE

Pericarditis occurs in 12 – 47% of living SLE patients[24]

Autopsy studies find a much higher prevalence of pericardial involvement ranging upto 61 – 100%.[31]

PREVALENCE OF PERICARDITIS

Study	No of patients	Clinical ascertainment (%)
Armas – Cruz et al[74]	108	12%
Griffith & Vural[31]	18	17%
Brigden et al[7]	60	43%
Harvey et al[78]	138	45.7%
Estes & christian[50]	150	19.3%
Kong et al[20]	30	47%
Badui et al[37]	100	25%
Sturfelt et al[75]	75	35%
Pistiner et al[65]	464	12%

PATHOLOGY

The histopathology in a case of pericarditis showed fibrosis, chronic inflammation with IgG, IgM and complement deposition on immunofluorescence.[13]

On immunofluorescence, IgG was present in a predominantly granular pattern around small pericardial vessels. Thus, Bidani and colleagues concluded that immune complex deposition was the cause of pericarditis.[14]

At autopsy, a diffuse or focal fibrinous pericarditis , often with many hematoxylin bodies, with or without effusion is found.[7]

Pericardial fluid is usually exudative, varying in amount from 100 to more than 1000cc.[10] WBC counts are in the 30,000 range, primarily neutrophils.

PREVALENCE OF PERICARDIAL EFFUSION

Study	No of Patients	Prevalence of effusion by Echocardiography
Ito et al[70]	48	46%
Chia et al[76]	21	24%
Crozier et al[80]	50	54%
Doherty et al[77]	50	42%
Sturfelt et al[75]	75	19%

Hunder et al found complement fixing immune complexes in the pericardial fluid of SLE patients.[69]

Anti DNA antibodies and low complement levels are seen in pericardial fluid.[16]

DIAGNOSIS

The diagnosis of pericarditis was based on the presence of a pericardial friction rub in 71%, ECG changes in 33% and on evidence of pericardial effusion in 50% of the patients.[24]

The diagnosis of pericarditis can be confirmed by ECG findings of elevated ST segments and tall T waves, or by cardiac echocardiogram findings of pericardial effusion or thickened pericardium.[67]

Serial ECGs may show a progression of changes in pericarditis. Initially a diffuse elevation of ST segments. This is followed by a lowering of ST segments back toward baseline and subsequent T wave inversion. In most cases, T waves then return to normal.[17]

TREATMENT

NSAIDS are the mainstay therapy[1.] Patients with pericardial tamponade may necessitate pericardiocentesis.[67]

2.MYOCARDITIS

Most myocarditis in SLE is subclinical. The clinical detection of myocarditis ranges from 3% to 15%.[67]

PREVALENCE OF MYOCARDITIS

Study	No of patients	Clinical Diagnosis(%)
Estes & Christian[50]	150	8%
Borenstein et al[96]	140	3.6%
Dubois & tuffanelli[97]	520	8%
Ropes[98]	128	10%
Badui et al[37]	100	14%
Godeau et al [11]	103	14.5%

Myocarditis should be considered in patients with tachycardia not due to fever, in patients with a third heart sound (S3), in patients with abnormal ECGs, in those with new murmurs or conduction disturbances, and in those with congestive cardiac failure.[10]

PATHOLOGY

Myocarditis in SLE is a complicated process, with arteritis or arteriopathy, not primary disease of the myocardial fibers.[18]

Immunofluorescence studies of endomyocardial biopsies reveal perivascular deposits of IgG and vascular deposits of C3.[19]

Kong et al found pathologic evidence of myocarditis – fibrinoid and collagenous degeneration, interstitial edema, necrosis, and cellular infiltration in 15 of 30 autopsies.[20]

DIAGNOSIS

The diagnosis of myocarditis can be made out by elevated Troponin levels, ECG abnormalities and supported by the finding of global hypokinesis on cardiac echocardiogram and confirmed by right ventricular endomyocardial biopsy.[21]

Hejtmancik et al made a clinical diagnosis of myocarditis in 21% of their patients based on

1.Cardiac enlargement

2.Conspicuous ventricular gallop rhythm

3.ECG abnormalities[24].

TREATMENT

Treatment with high dose intravenous methylprednisolone, followed by high dose intravenous or oral corticosteroid maintenance therapy is indicated.

The addition of intravenous pulse cyclophosphamide , in refractory cases, may be helpful.[67]

Efficacy of therapy can be assessed by serial echocardiographic studies or right ventricular endomyocardial biopsies.[23]

3. LEFT VENTRICULAR DYSFUNCTION

Echocardiographic studies consistently show that 4-71% of SLE patients have some degree of left ventricular dysfunction. [12, 24]

PREVALENCE OF LEFT VENTRICULAR DYSFUNCTION

Study	No of patients	Frequency(%)
Chia et al[76]	21	71%
Roldan et al[35]	54	20%
Leung et al[12]	75	5%
Doherty et al[77]	50	10%

SLE patients may have systolic dysfunction that only becomes apparent with exercise.[25] Diastolic dysfunction although subclinical is found more consistently.[26, 27]

Giunta et al found that disease duration was longer in patients with diastolic dysfunction.[27]similarly Enomoto et al found that diastolic function deteriorated progressively with age.[26]

PATHOLOGY

The study of Strauer et al found multiple abnormalities in SLE patients including,

1. Increased end diastolic pressures.
2. Decreased contractility.
3. Decreased left ventricular ejection fraction.
4. Increased left ventricular stiffness.
5. Reduction of coronary vascular reserve.[28]

Corticosteroid therapy could contribute to ventricular dysfunction through multiple mechanisms, including fatty infiltration.[54] A second potential factor is hypertension aggravated by corticosteroids.[86]

DIAGNOSIS

The diagnosis of subtle degrees of left ventricular systolic or diastolic dysfunction is made echocardiographically.[67]

Left ventricular systolic function can be evaluated by the left ventricular ejection fraction[67].

Diastolic function can be determined by the diastolic descent rate of the anterior mitral leaflet, The ratio of mean systolic velocity to mean diastolic velocity to mean diastolic velocity in the left ventricular posterior wall.[26]

TREATMENT

Ventricular dysfunction that progressively worsens in inactive patients might be best addressed by aggressive risk factor modification and pharmacologic therapy.[71] However ventricular function worsening with active lupus might improve with corticosteroid treatment.[70]

4.VALVULAR DISEASE

The prevalence of valvular disease in SLE is very high.[12, 24, 29]

PREVALENCE OF VALVULAR DISEASE

Study	No of patients	Valvular disease	Frequency(%)
Leung et al[12]	75	Valve thickening-gross	8%
		Valve thickening-focal	12%
		Mitral regurgitation	25%
		Aortic regurgitation	8%
Sturfelt et al[75]	75	Valve thickening	45%
		Mitral regurgitation	39%
		Aortic regurgitation	13%
		Vegetations	4%
Guinta et al[27]	75	Valve thickening	12%
		Vegetations	4%
Galve et al[84]	74	Mitral valve thickening	12%
		Vegetations	9%
Badui et al[37]	100	Valvular disease	9%

PATHOLOGY

The mitral valve is affected most often, followed by the aortic valve. Mitral and aortic regurgitation are the most common findings, with stenotic lesions being very rare.[67]

The typical valvular and mural endocarditis lesions, which are verrucous, occur as a single vegetation or as mulberry like clusters. When occuring on valves the vegetations are often on the ventricular surface, near, but not distorting, the line of closure.[30]

In the corticosteroid era, valvular vegetations are found less frequently.[67]

Shearn found that none of the 11 patients who received corticosteroids had verrucous endocarditis.[10]

The original histologic description of Libman-Sacks endocarditis emphasized the multiplication of endothelial cells, proliferation of Anitschow myocytes, and infiltration of mononuclear cells in the valve ring and valve base, especially the valve pocket.[32, 33]

Immunofluorescence showed immunoglobulin and complement deposition in the walls of small junctional vessels in the inner zone of neovascularization, suggesting that circulating immune complexes were critical in the development of the vegetations.[30]

Galve et al found that patients with Libman-sacks endocarditis were younger, had shorter disease duration, and had received less corticosteroid therapy than those with thickened valves.[84]

CLINICAL FEATURES

Shearn found that systolic murmurs occurred in 70% of SLE patients. Diastolic murmurs occur in only 4% of SLE patients.[74,]

Griffith and vural heard murmurs in only two of six patients with Libman-Sacks endocarditis, and vice versa, found Libman-Sacks endocarditis in only two of seven patients with systolic murmurs.[31]

DIAGNOSIS

Transesophageal echocardiogram is the modality of choice in terms of sensitivity in detecting valvular disease due to lupus.[34, 35]

TREATMENT

SLE patients with large , sterile vegetations should be anticoagulated to lessen embolic complications. High dose corticosteroids for 4 to 6 weeks to shrink vegetations is controversial.[36]

5.ARRYTHMIAS AND CONDUCTION DISTURBANCES

The strongest association of SLE with conduction disturbance is congenital heart block, usually in the setting of maternal anti-RO and anti-La.[38,39]

PREVALENCE

Approximately 10% of adult SLE patients have conduction disturbances.[11, 37]

Sinus tachycardia is found in 6 – 100% of patients[24,]

Arrhythmias are found more commonly in SLE patients with pericarditis and myocarditis.[67]

PREVALENCE OF SINUS TACHYCARDIA

Study	Frequency (%)
Badui et al[37]	11 %
Griffith & Vural[31]	100%
Hejtmancik et al[24]	50%

PATHOLOGY

Autopsy studies of SLE patients have found arteritis of the sinus node, vascular occlusion, vasculopathy, and fibroblastic replacement of the sinoatrial and atrioventricular nodes.[40]

DIAGNOSIS

Accurate ascertainment of arrhythmias requires continuous ECG monitoring.[67]

TREATMENT

SLE patients with life-threatening conduction defects can be treated with permanent pacemakers.[41]

6.CORONARY ARTERITIS.

Coronary arteritis is extremely rare in SLE. In some cases, it has been found at autopsy, with no clinical correlate during life.[67]

PREVALENCE

There are few studies that allow any estimate of the prevalence of coronary arteritis.[7, 24, 18, 20].

In one study in 1960, 6 of the 16 patients were found to have arteritis at biopsy.[24]

CORONARY ARTERITIS

Case report or series	No of patients	Age at diagnosis
Homcy et al[44]	3	27, 34, 21
Bonfiglio[42]	1	16
Korbet et al[43]	1	26
Heibel et al[45]	1	45
Hejtmancik et al[24]	6	-
Brigden et al[7]	2	-
Kong et al[20]	1	-
Simon et al[18]	1	25

CLINICAL FEATURES

The most common clinical presentation is angina, myocardial infarction, or both in a child or young adult.[67]

PATHOLOGY

Histopathology demonstrates transmural vasculitis.[42] Immunofluorescence studies demonstrate immunoglobulin and complement deposition in coronary arteritis.[43]

DIAGNOSIS

It is often difficult to distinguish coronary arteritis from accelerated atherosclerosis. Serial coronary angiography has been proposed as the most useful diagnostic modality.[44, 45] Arteritis is suggested when coronary aneurysms are found, if there are smooth focal lesions, or if there are rapidly developing stenoses.[44, 45]

TREATMENT

The differentiation of coronary arteritis from atherosclerosis is essential for appropriate management. Coronary artery bypass surgery, angioplasty, or stent placement would be contraindicated in patients with coronary arteritis.[67]

Case reports suggest that corticosteroid therapy can have rapid benefit in patients with coronary arteritis.[67]

7. PULMONARY HYPERTENSION

Pulmonary hypertension is unusual in SLE patients. Earlier studies, which determined the prevalence clinically, found a cumulative frequency of only 2-9%.[67]

PREVALENCE OF PULMONARY HYPERTENSION

Study	No of patients	Frequency(%)
Brigden et al[7]	60	3%
Perez & Kramer[46]	43	9%
Badui et al [37]	100	9%
Crozier et al[80]	50	2%
Hejtmancik et al[24]	142	1%
Quismorio et al[82]	400	1%
Simonson et al[83]	36	14%
Leung et al[12]	75	1%

CLINICAL FEATURES

It is usually asymptomatic, discovered on a screening ECHO Doppler.

Rare SLE patients will present with chest pain, dyspnea, or even pedal edema and be found to have pulmonary hypertension.[67]

PATHOLOGY

Several lines of evidence suggest that pulmonary hypertension may be a complication of pulmonary artery vasospasm.[67]

Raynaud's phenomenon is more common in SLE patients with pulmonary hypertension.[46, 47]

In one series of SLE patients, those with pulmonary hypertension by Doppler had a shorter duration of SLE and corticosteroid therapy and a higher prevalence of Raynaud's phenomenon.[82]

DIAGNOSIS

Diagnosis is best made by Doppler echocardiography. Doppler echocardiography has a close correlation with simultaneous right heart catheterization measurement of pulmonary artery pressures. Owing to invasive nature right heart catheterization is more appropriately reserved for symptomatic patients.[67]

TREATMENT

Treatment is now available for severe pulmonary hypertension with the advent of continuous intravenous prostracyclin and its analogs.[48] Patients with severe pulmonary hypertension should be anticoagulated.[49]

8. HYPERTENSION

Although earlier studies did not find a high prevalence of hypertension in SLE patients because of shortened survival, more recent studies have found a high frequency of up to 50%.[67]

Several studies found that hypertension was more common in those with underlying lupus nephropathy.[10]

PREVALENCE OF HYPERTENSION

Study	Prevalence(%)
Harvey et al[78]	14%
Shearn[10]	32%
Brigden et al[7]	44%
Kong et al[20]	53%
Hejmancik et al[24]	22%
Okado & Shiokawa[85]	44%
Budman & Steinberg[79]	45%
Doherty et al[77]	50%
Crozier et al[80]	14%
Schioppati & Remuzzi[81]	40%

86% of SLE patients with hypertension had lupus nephritis in the series of Estes and Christian.[50]

Pollack and Kant found a correlation of mean diastolic blood pressure and increasing renal damage.[51]

Hypertension is likely to develop or worsen when patients with lupus nephropathy are given corticosteroids.[52]

When examined the relationship of prednisone and blood pressure using the Hopkins Lupus cohort database, it is found that an increase in Prednisone dose of 10mg led to an increase in mean arterial pressure, adjusting for all other factors that affect blood pressure.[66]

TREATMENT

As many hypertensive patients with SLE have underlying renal disease, long term benefit of ACE inhibitors is lessening of renal scarring.[53]

ACE inhibitors are well tolerated in SLE, although an occasional patient may develop an ACE inhibitor induced chronic cough.[67]

9. CORONARY ATHEROSCLEROSIS IN SLE.

coronary atherosclerosis is a clinical conundrum of the modern era of lupus management[67].

Myocardial infarction was not common in early autopsy series, but was a major feature of the Bulkley and Roberts and subsequent autopsy series.[54]

Patients usually present in early 40s with angina, myocardial infarction or sudden death. However, patients have presented in their early 20s with coronary atherosclerosis.[55]

Clinically, the patient may present with anginal pain, stable or unstable angina, acute myocardial infarction or heart failure. The differential diagnosis includes coronary arteritis, thrombosis secondary to antiphospholipid antibody syndrome or coronary vasospasm.[67]

PATHOGENESIS

IMMUNE COMPLEX OR ARTERITIS

Although coronary arteritis is rarely detected ante mortem autopsy studies have detected it frequently.[31, 54]

Immune complexes from lupus sera accelerated uptake of cholesterol by smooth muscle cells.[56] vascular injury, through immune complexes, followed by exposure to atherosclerotic risk factors, can lead to atherosclerosis.[57]

Both arteritis and atherosclerosis have been found within a single patient, suggesting that arteritis might have predisposed to the later development of atherosclerosis.[24]

SLE Patients treated with corticosteroids had less intimal proliferation in their coronary vessels, suggesting that suppression of arteritis might lead to less atherosclerosis.[58]

ANTI-PHOSPHOLIPID ANTIBODIES

Anti-phospholipid antibodies contribute to coronary artery disease through thrombosis[59] or vasculopathy.[60]

Anti-phospholipid antibodies function as antibodies against oxidized lipoproteins, by which they contribute to atherosclerosis.[61]

Beta 2 glycoprotein 1, an important control against atherosclerosis is perturbed by anti-phospholipid antibodies.[62]

PREVALENCE OF CORONARY ARTERY DISEASE

Prospective studies have found, using the clinical detection of angina, myocardial infarction, or both, frequencies on the order of 7-9%.[11, 12]

Study	No of patients	Modality	Frequency(%)
Kong et al[20]	30	Myocardial infarction	7%
Bulkley&	36	>50% narrowing	22%
Roberts[54]		Myocardial infarction	11%
Sturfelt et al[75]	75	Myocardial infarction	9%
		Exercise induced Ischemia	11%
Hetjmancik[24]	142	Angina in 6 patients, EKG changes in 4	4.9%
Badui et al[37]	100	-	16%
Urowitz et al[100]	81	Angina, Myocardial infarction	7.4%
Griffith & Vural[31]	11	-	45%
Bidani et al[14]	10	-	10%

RISK FACTORS FOR CAHD IN SLE

Some of the risk factor could be due to SLE . Hypertension, for example is more prevalent in SLE patients with renal disease.[74, 50]

Prolonged corticosteroid therapy could precipitate atherosclerosis indirectly by hypertension, hypercholesterolemia, hypertriglyceridemia, diabetes mellitus, obesity and hyperhomocysteinemia or directly, via vascular injury[87].

HYPERTENSION

Hypertension is very prevalent in SLE patients and is aggravated by corticosteroids.[66]

HYPERLIPIDEMIA

Hyperlipidemia in SLE has two major patterns

FIRST PATTERN

Low HDL

Low apoprotein A1

Elevated VLDL

Elevated triglycerides

This pattern is seen in active disease.[88]

SECOND PATTERN

High triglycerides

High LDL

High total cholesterol

This pattern occurs in SLE patients on corticosteroids.[89]

HYPERHOMOCYSTEINEMIA

Homocysteine is an amino acid that has a direct toxic effect on endothelium[90] and indirect effects, including promotion of vascular smooth muscle proliferation and an inhibitory effect on endothelial cell growth[91].

In the Hopkins Lupus cohort study, 15% of the 337 SLE patients had elevated homocysteine.[67]

Raised homocysteine levels are associated with CAHD, stroke and arterial thrombosis.[92]

PREVALENCE OF CAHD RISK FACTORS[67]

Risk factor	Prevalence (%)
Family history	41%
Hypertension	48%
Hypercholesterolemia	56%
Obesity	38%
Smoking	56%
Sedentary lifestyle	70%
Diabetes	7%
Homocysteine	15%

DIAGNOSIS

Coronary Angiography remains the gold standard for the diagnosis of coronary artery disease.[67]

Rest and perfusion myocardial single emission Computed Tomography (SPECT) scans are currently one of the most sensitive and specific means of assessing the presence of atherosclerosis.[93]

TREATMENT

The acute management of an SLE patient with myocardial infarction is similar to that of a non SLE patient.[67]

High dose corticosteroid therapy should only be given at the time of myocardial infarction if there is proof of arteritis by angiogram or a very high suspicion based on extra cardiac active lupus, because of the possible adverse effect it may have in causing marked scar thinning[94].

FUTURE TREATMENT

The presence of macrophages and activated T cells in atherosclerotic plaques suggest that immunity plays a role in atherosclerotic progression. CD40 – CD40 ligand interactions may be important in the development of atherosclerosis[67].

In atherosclerotic plaques, triggering of CD40L on T cells leads to regulation of T-cell expansion and cytokine production[95].

Anti – CD40 ligand, therapy already being considered as a novel way to treat SLE may have the additional benefit of retarding atherosclerosis[67].

CARDIOVASCULAR MORTALITTY

Urowitz and colleagues drew attention to the bimodal pattern of mortality in SLE, with early deaths due to active disease and infection and later deaths due to cardiovascular disease.[63]

CARDIOVASCULAR MORTALITY

Study	Death due to cardiovascular disease (%)
Urowitz et al[100]	45%
Karsh et al[73]	25%
Wallace et al[64]	20%
Rosner et al[87]	3%
Pistiner et al[65]	15%

In the study of causes of death in 144 SLE patients, cardiovascular disease was the third leading cause of death[67].

With better survival of SLE patients, both the morbidity and mortality from accelerated atherosclerosis tend to increase.[50, 65, 66]

MATERIALS AND METHODS

The study was conducted in Thanjavur Medical College Hospital, Thanjavur, Tamilnadu. The study was conducted in the Department of Internal Medicine. The study period extended between June 2009 and October 2010. It was a carefully selected study population of SLE based on 1997 UPDATE OF THE 1982 AMERICAN COLLEGE OF RHEUMATOLOGY CLASSIFICATION CRITERIA FOR SLE.

The patients were selected on the basis of inclusion and exclusion criteria and cardiac evaluation was done.

INCLUSION CRITERIA

All registered cases of SLE (diagnosed on the basis of 1997 UPDATE OF THE 1982 AMERICAN COLLEGE OF RHEUMATOLOGY CLASSIFICATION CRITERIA FOR SLE) attending Nephrology, Cardiology and Medical departments are included for the study

EXCLUSION CRITERIA

Tuberculosis

Rheumatic heart disease

Uremia

Intake of drugs or conditions other than SLE, producing positive ANA.

A profoma was drafted including the details about the presenting illness and all patients were subjected to routine physical examination including detailed cardiovascular examination.

Routine blood investigations like complete hemogram, blood sugar, blood urea, serum creatinine, serum electrolytes and erythrocyte sedimentation rate were done.

Complete urine analysis including urine albumin, deposits and 24 hours urinary protein were done.

ANA and dsDNA tests were done for all patients. Anti phospholipid antibody test was done for patients who gave history of fetal wastage.

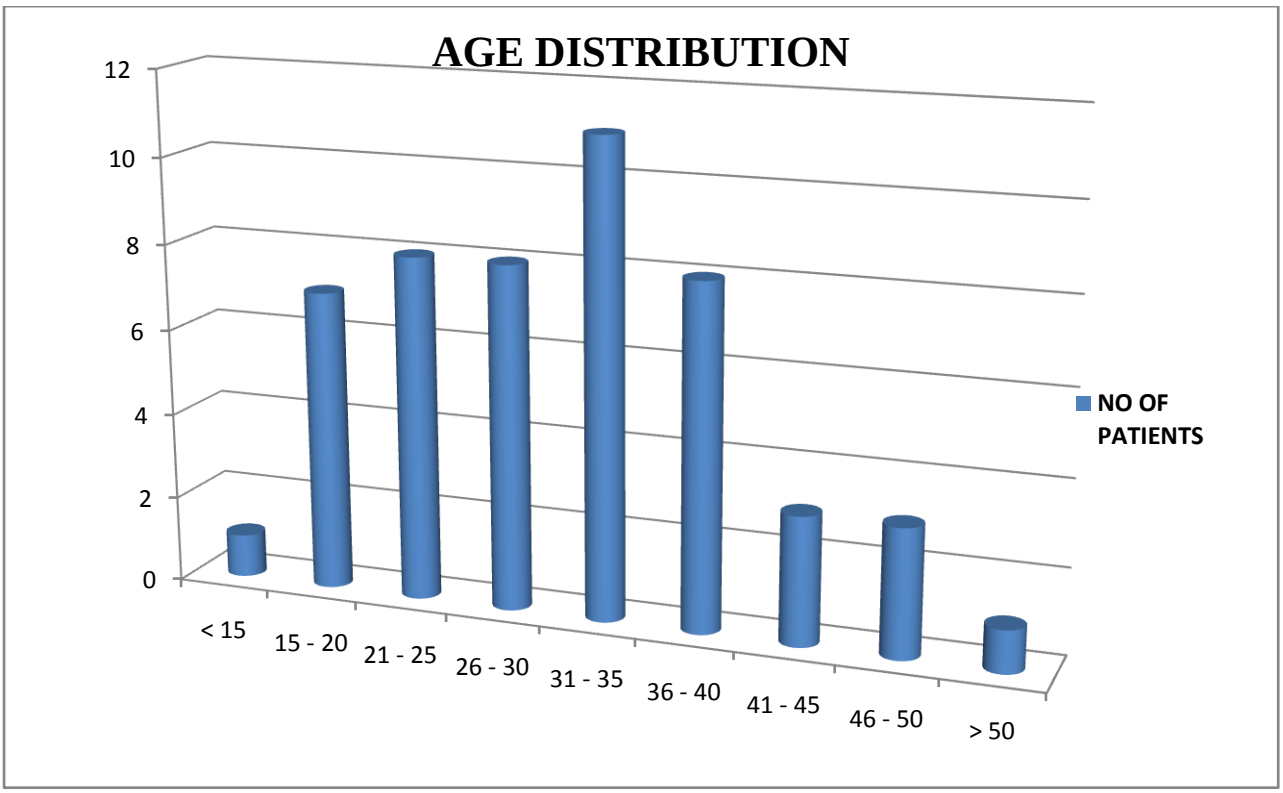
After taking ECG and X ray chest, all patients were subjected to Echocardiography.

Patient Name : Miss. Gomathi
Age: 22 Years
IP Number: 1088786

A SYSTEMIC LUPUS ERYTHEMATOSUS PATIENT WITH MALAR RASH AND ALOPECIA

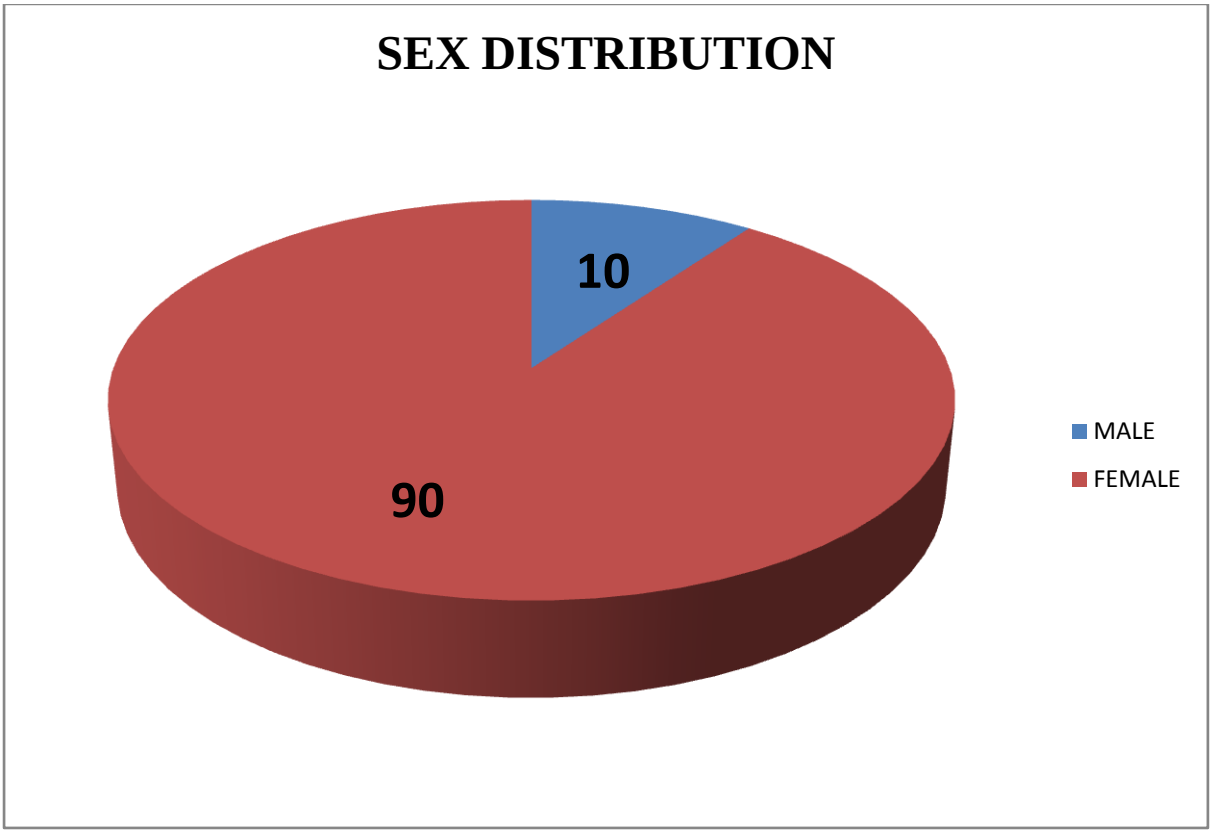
AGE DISTRIBUTION

AGE	NO OF PATIENTS	PERCENTAGE
< 15	1	2%
15 - 20	7	14%
21 - 25	8	16%
26 - 30	8	16%
31 - 35	11	22%
36 - 40	8	16%
41 - 45	3	6%
46 - 50	3	6%
>50	1	2%



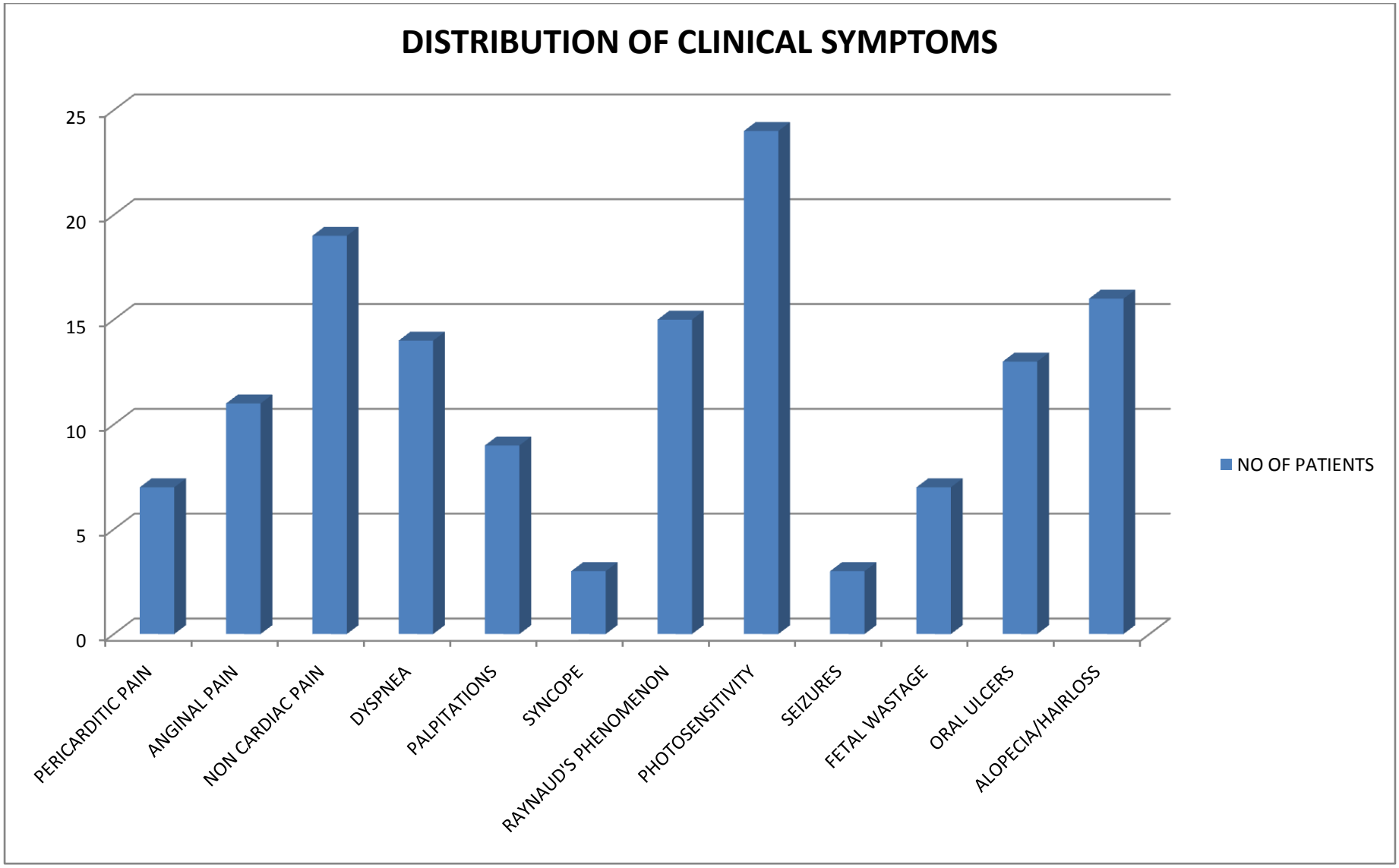
SEX DISTRIBUTION

SEX	NO OF PATIENTS	PERCENTAGE
MALE	5	10%
FEMALE	45	90%



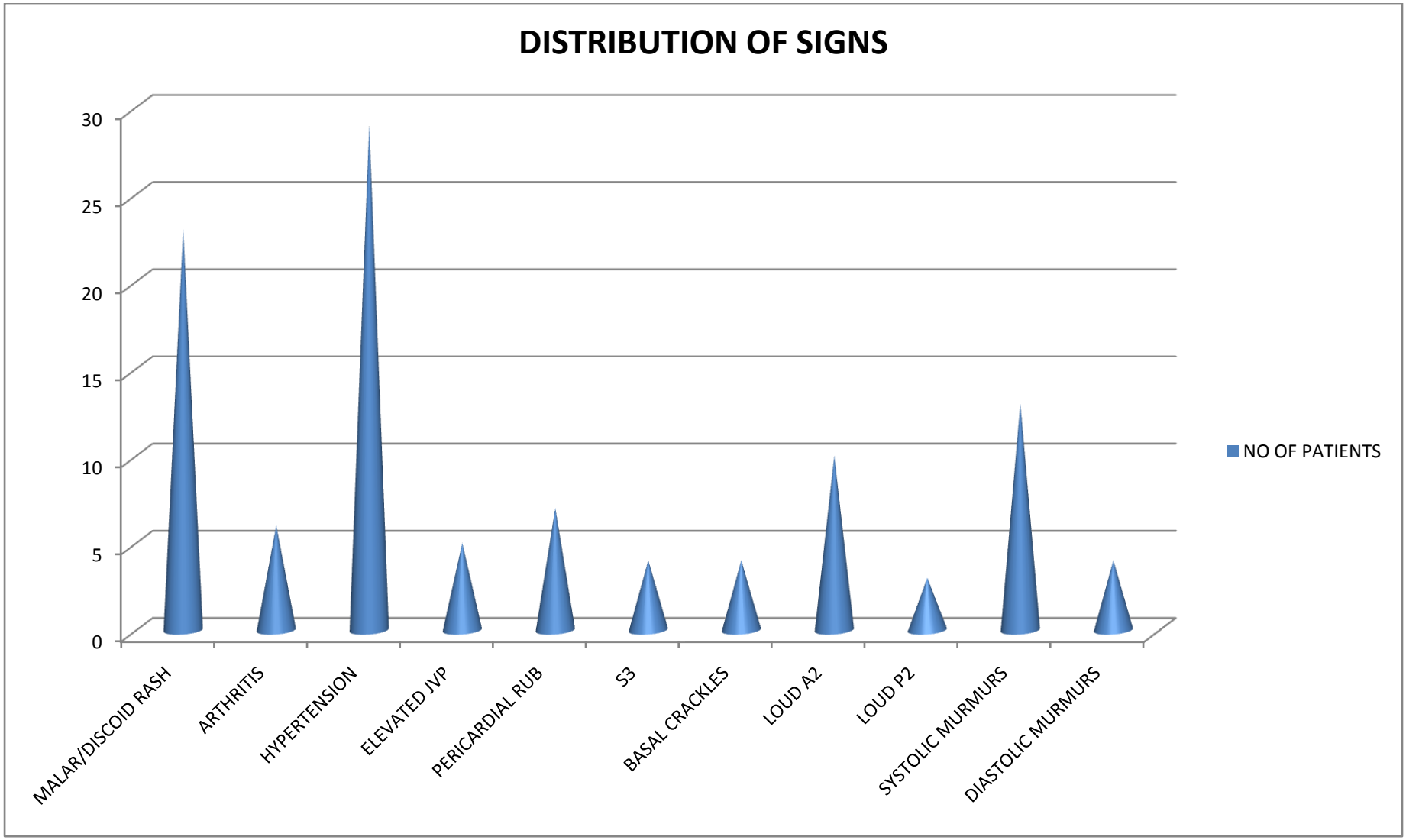
DISTRIBUTION OF CLINICAL SYMPTOMS

SYMPTOMS	NO OF PATIENTS	PERCENTAGE
CHEST PAIN	37	74%
PERICARDITIC	7	14%
ANGINAL	11	22%
NON CARDIAC	19	38%
DYSPNEA	14	28%
PALPITATIONS	9	18%
SYNCOPE	3	6%
RAYNAUD'S PHENOMENON	15	30%
PHOTO SENSITIVITY	24	48%
SEIZURES	3	6%
FETAL WASTAGE	7	14%
ORAL ULCERS	13	26%
ALOPECIA/ HAIR LOSS	16	32%


DISTRIBUTION OF CLINICAL SYMPTOMS
25
20
15
10
5
0
PERICARDITIC PAIN
ANGINAL PAIN
NON CARDIAC PAIN
DYSPNEA
PALPITATIONS
SYNCOPE
RAYNAUD'S PHENOMENON
PHOTOSENSITIVITY
SEIZURES
FETAL WASTAGE
ORAL ULCERS
ALOPECIA/HAIRLOSS
NO OF PATIENTS

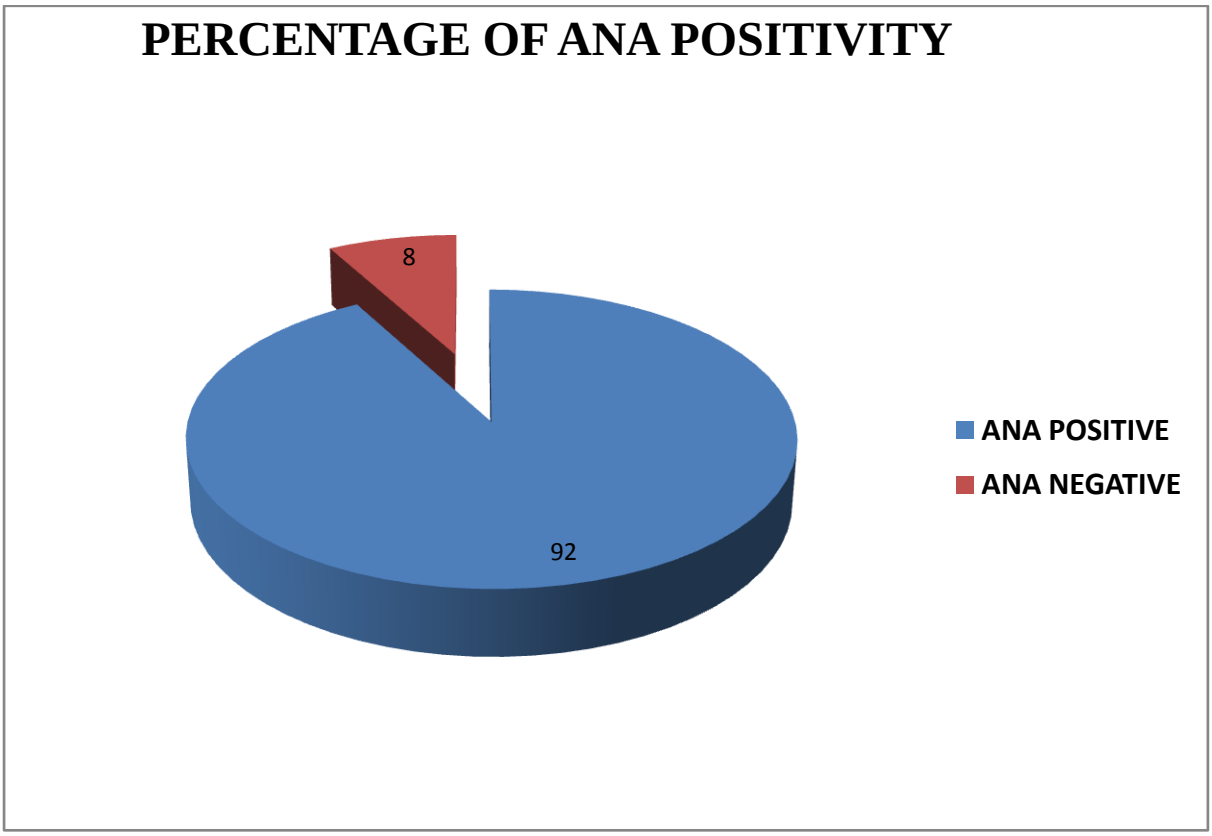
DISTRIBUTION OF SIGNS

SIGNS	NO OF PATIENTS	PERCENTAGE
MALAR/DISCOID RASH	23	46%
ARTHRITIS	6	12%
HYPERTENSION	29	58%
ELEVATED JVP	5	10%
PERICARDIAL RUB	7	14%
S3	4	8%
BASAL CRACKLES	4	8%
LOUD A2	10	20%
LOUD P2	3	6%
MURMURS		
SYSTOLIC	13	26%
DIASTOLIC	4	8%


DISTRIBUTION OF SIGNS
30
25
20
15
10
5
0
MALAR/DISCOID RASH
ARTHRITIS
HYPERTENSION
ELEVATED JVP
PERICARDIAL RUB
S3
BASAL CRACKLES
LOUD A2
LOUD P2
SYSTOLIC MURMURS
DIASTOLIC MURMURS
NO OF PATIENTS

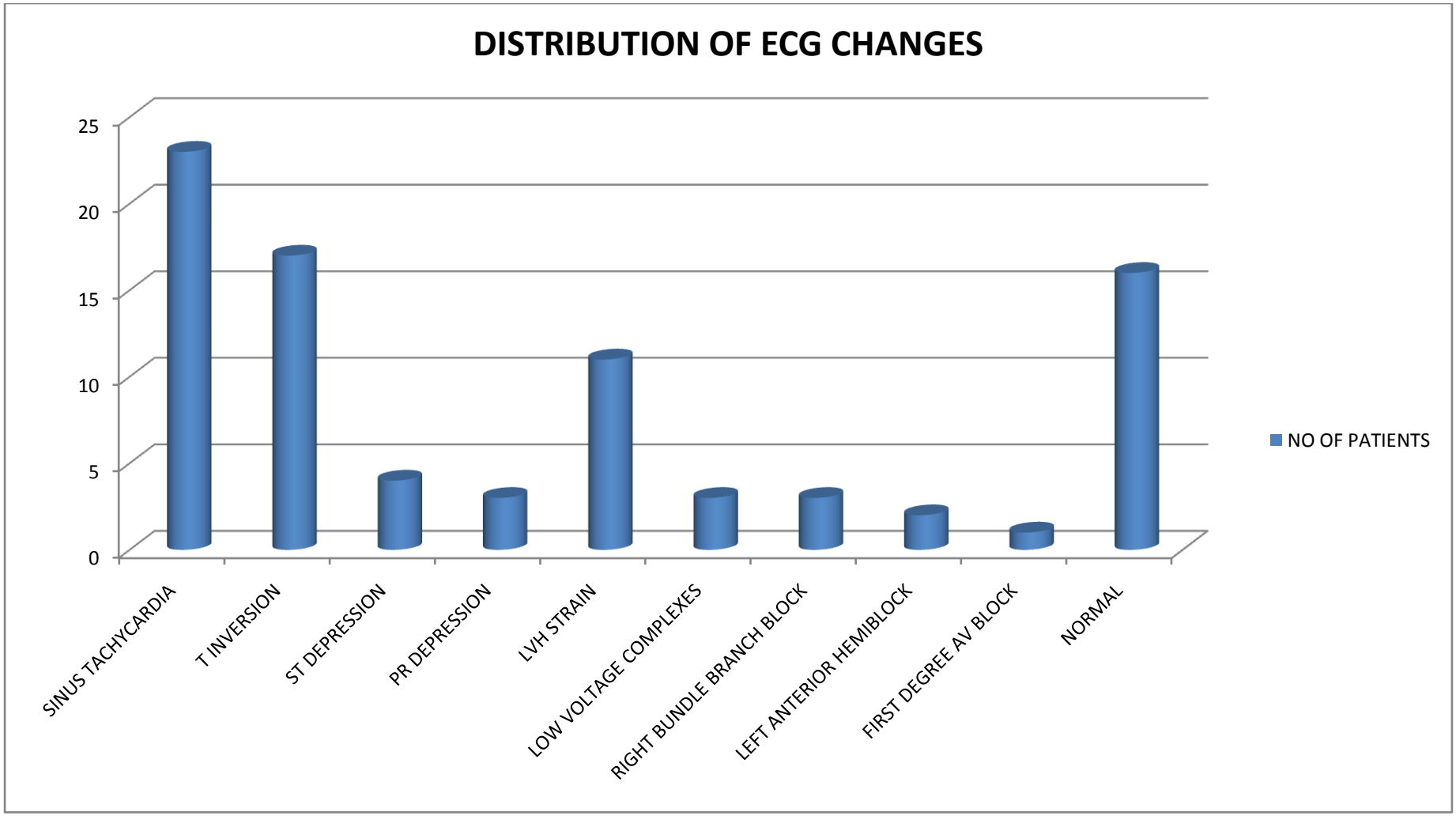
ANTI NUCLEAR ANTIBODY POSITIVITY

ANTI NUCLEAR ANTIBODY	NO OF PATIENTS	PERCENTAGE
POSITIVE	46	92%
NEGATIVE	4	8%



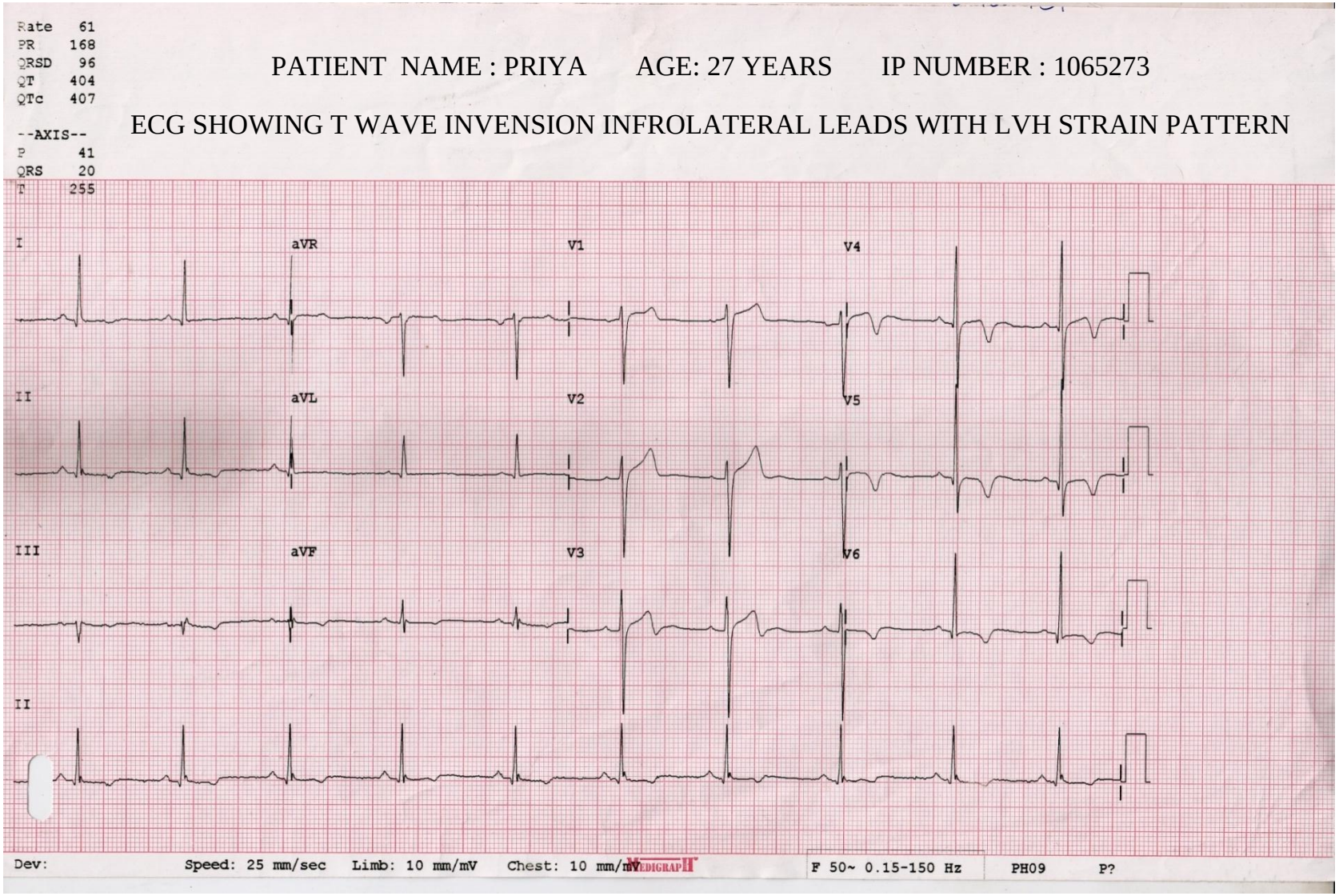
DISTRIBUTION OF ECG CHANGES

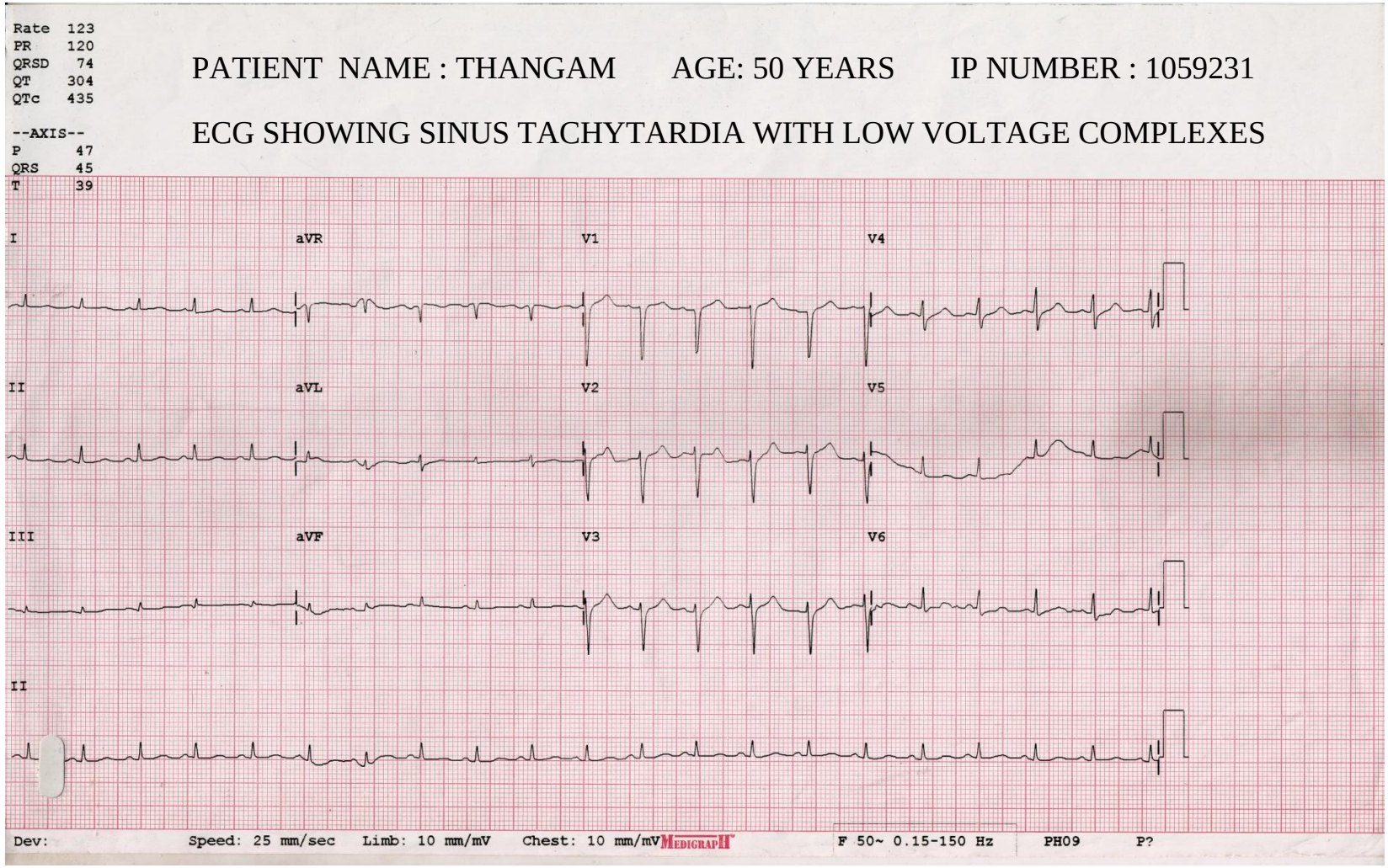
ECG CHANGES	NO OF PATIENTS	PERCENTAGE
SINUS TACHYCARDIA	23	46%
T INVERSION	17	34%
ST DEPRESSION	4	8%
PR DEPRESSION	3	6%
LVH STRAIN	11	22%
LOW VOLTAGE COMPLEXES	3	6%
RIGHT BUNDLE BRANCH BLOCK	3	6%
LEFT ANTERIOR HEMIBLOCK	2	4%
FIRST DEGREE AVBLOCK	1	2%
NORMAL	16	32%


DISTRIBUTION OF ECG CHANGES
25
20
15
10
5
0
SINUS TACHYCARDIA
T INVERSION
ST DEPRESSION
PR DEPRESSION
LVH STRAIN
LOW VOLTAGE COMPLEXES
RIGHT BUNDLE BRANCH BLOCK
LEFT ANTERIOR HEMIBLOCK
FIRST DEGREE AV BLOCK
NORMAL
NO OF PATIENTS

PATIENT NAME : PRIYA AGE: 27 YEARS IP NUMBER : 1065273

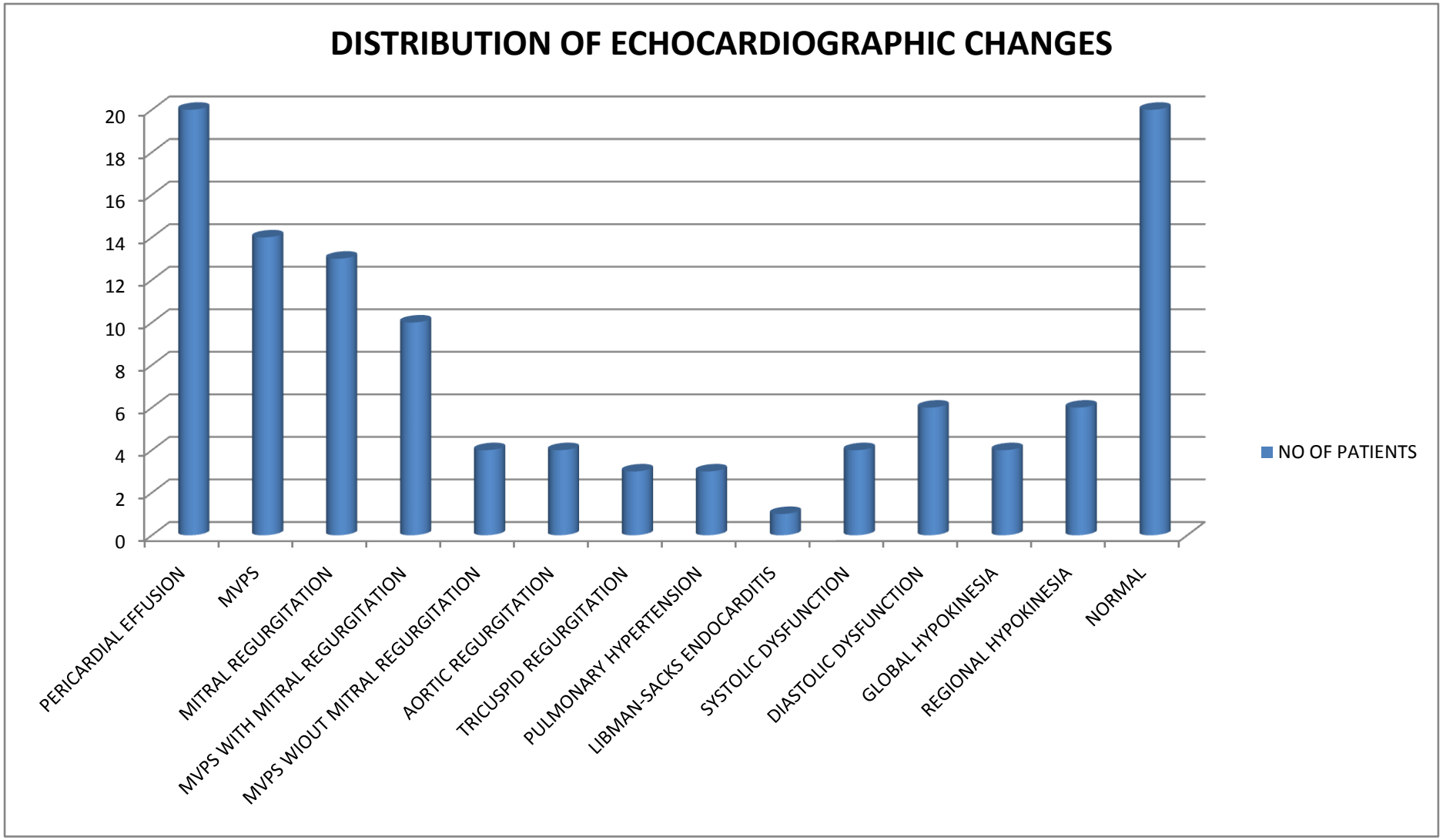
ECG SHOWING T WAVE INVENSION INFROLATERAL LEADS WITH LVH STRAIN PATTERN




Rate 123
PR 120
QRSD 74
QT 304
QTc 435
--AXIS--
P 47
QRS 45
T 39
PATIENT NAME : THANGAM AGE: 50 YEARS IP NUMBER : 1059231
ECG SHOWING SINUS TACHYTARDIA WITH LOW VOLTAGE COMPLEXES
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Dev:
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10 mm/mV
MEDIGRAPH
F 50~ 0.15-150 Hz
PH09
P?

DISTRIBUTION OF ECHOCARDIOGRAPHIC CHANGES

ECHO FINDINGS	NO OF PATIENTS	PERCENTAGE
PERICARDIAL EFFUSION	20	40%
MVPS	14	28%
MITRAL REGURGITATION	13	26%
MVPS WITH MITRAL REGURGITATION	10	20%
MVPS WITH OUT MITRAL REGURGITATION	4	8%
AORTIC REGURGITATION	4	8%
TRICUSPID REGURGITATION	3	6%
PULMONARY HYPERTENSION	3	6%
LIBMAN – SACKS ENDOCARDITIS	1	2%
SYSTOLIC DYSFUNCTION	4	8%
DIASTOLIC DYSFUNCTION	6	12%
HYPOKINESIS GLOBAL REGIONAL	10 4 6	20% 8% 12%
NORMAL	20	40%


DISTRIBUTION OF ECHOCARDIOGRAPHIC CHANGES
20
18
16
14
12
10
8
6
4
2
0
PERICARDIAL EFFUSION
MVPS
MITRAL REGURGITATION
MVPS WITH MITRAL REGURGITATION
MVPS WIOUT MITRAL REGURGITATION
AORTIC REGURGITATION
TRICUSPID REGURGITATION
PULMONARY HYPERTENSION
LIBMAN-SACKS ENDOCARDITIS
SYSTOLIC DYSFUNCTION
DIASTOLIC DYSFUNCTION
GLOBAL HYPOKINESIA
REGIONAL HYPOKINESIA
NORMAL
NO OF PATIENTS

RESULTS AND OBSERVATIONS

The study population included 50 Systemic Lupus Erythematosus patients, of whom 45 patients are females and 5 are males, with majority of patients distributed between the age group of 20 to 40 years. The lowest age is 13 years and the highest age is 60 years. The maximum prevalence of SLE is in the age group of 31 to 35 years.

Out of 50 Systemic Lupus Erythematosus patients 46 patients are positive for Anti Nuclear Antibody and 4 patients are negative for Anti Nuclear Antibody.

37 out of 50 patients had chest pain, of whom11 patients had anginal type of pain, 7 had pericarditis type of pain, and 19 had non specific chest pain.

Among 50 patients, 14 patients had dyspnea, 9 patients had palpitations and 3 patients had syncope

Cutaneous photosensitivity was noted in 24 patients, 23 patients had Malar / Discoid rash, 15 patients had positive Raynaud’s phenomenon.

Out of 50 patients 7 female patients had previous fetal wastage, all are positive for Antiphospholipid antibodies and 3 patients had history of seizures.

13 patients had oral ulcers, 16 patients had alopecia / hair loss, 6 patients had Arthritis and 44 patients had Lupus nephropathy.

Systemic hypertension was found in 29 patients and all patients had associated Lupus nephropathy

Of the 50 SLE patients, 5 patients had elevated JVP , 7 patients had pericardial rub, Loud aortic component of second heart sound was found in 10 patients, loud pulmonary component of second heart sound was found in 3 patients, 4 patients had third heart sound on auscultation. Systolic murmurs were found in 13 patients and 4 patients had diastolic murmurs.

Bilateral basal crackles was found in 4 patients due to left ventricular dysfunction, in these patients the x ray chest taken ruled out the possibility of respiratory disease to prove basal crackles are due to cardiac disease.

ECG CHANGES

Normal ECG was found in 16 SLE patients, 23 patients had sinus tachycardia. T wave inversion was found in 17 patients , 11 patients had LVH strain, 4 patients showed ST depression and 3 patients showed PR depression in their ECG. Low voltage complexes was found in 3 patients, 6 patients had conduction disturbances in their ECG, out of which 3 patients had Right Bundle Branch Block, 2 patients had Left Anterior Hemi Block and 1 patient had first degree AV block.

X RAY

Out of 50 SLE patients 9 patients showed cardiomegaly (Cardio Thoracic ratio > 0.5) in their X – rays.

ECHOCARDIOGRAPHIC FINDINGS

Among 50 patients 20 patients had normal Echocardiography

20 out of 50 patients had pericardial effusion, which is the commonest echocardiographic finding. Of these 20 patients ,15 had mild pericardial effusion and 5 had moderate pericardial effusion , massive pericardial effusion so as to cause cardiac tamponade was found in no patient.

14 patients had mitral valve prolapse, 13 patients had mitral regurgitation and 10 patients had MVPS associated with mitral regurgitation .

Out of 50 patients 4 patients had Aortic regurgitation , 3 patients had tricuspid regurgitation , pulmonary hypertension was found in 3 patients and 1 patient had Libman-sacks endocarditis

10 patients had left ventricular dysfunction of whom 4 patients had systolic dysfunction and 6 patients had diastolic dysfunction.

Out of 50 SLE patients 4 patients had global hypokinesia and 6 patients had regional hypokinesia in their ECHO

DISCUSSION

PERICARDITIS

In this study Pericarditis is the most common cardiac manifestation.

Armas – Cruz et al ,Brigden et al and Kong et al showed that pericarditis is the most common cardiac manifestation in SLE patients ranging from 12 – 47% [7,20,74], prevalence of pericarditis in our study is 40% which tally with the literature finding.

MITRAL REGURGITATION

Valvular heart disease is the second most common cardiac manifestation in SLE next to pericarditis

Leung et al and Sturfelt et al showed that 25 – 39% of SLE cases had mitral regurgitation.[12, 75] Here, in our study, 13 out of 50 SLE patients had mitral regurgitation accounting for 26% . This finding also coincides with the literature quoted.

AORTIC REGURGITATION

Leung et al and Sturfelt et al showed that Aortic regurgitation occurs in 8 – 13% of SLE patients.[12, 75]

In this study Aortic regurjutation is observed in 4 patients accounting for 8%. This finding also tally with the literature.

LIBMAN – SACKS ENDOCARDITIS

According to studies conducted by Sturfelt et al, Giunta et al and Galve et al Libman – Sacks endocarditis occurs in 4 – 9% of SLE patients.[75,84] Libman-Sacks lesions have been noted in 25% to 100% and infective endocarditis in 1.1 to 4.9% of clinical and autopsy studies done by Doherty NE.[101] In our study endocarditis is observed echocardiographically in 1 patient accounting for 2% , the prevalence of endocarditis is lesser when compared to the literature. The lower prevalence could be due to the use of steroids , as majority of the patients are receiving treatment for associated Lupus nephropathy(88%).

ARRHYTHMIAS AND CONDUCTION DISTURBANCES

According to Badui et al and Griffith and Vural et al sinus tachycardia occurs in 11 to 100%.[31, 37] In our study sinus tachycardia occurred in 23 patients accounting for 46%. Approximately 10% of SLE patients have conduction disturbances.[11, 32, 85] In this study 6 patients had conduction disturbances accounting for 12%. Of whom 3 patients had Right Bundle Branch Block, 2 patients had Left Anterior Hemi Block and 1 patient had first degree AV block.

HYPERTENSION

Harvey et al, Kong et al, Budman and Steinberg et al, Doherty et al , Crozier et al, Shieppati and Remuzzi et al showed that hypertension is seen in SLE patients of about 14 – 53 %[20, 77, 78, 79, 80, 81]

In this study hypertension is observed in 29 patients which is about 58 %. This higher prevalence of hypertension is due to associated lupus nephropathy(88%) and the patients are receiving steroids for it.

PULMONARY HYPERTENSION

Hejtmancik et al, Perez and Kramer et al, Quismorio et al, Surfeit et al and Simonson et al showed that pulmonary hypertension occurs in 1 – 9% of patients with SLE.[24, 45, 75, 82, 83] In this study pulmonary hypertension is found in 3 patients accounting for 6%. This finding also tally with the literature quoted.

LEFT VENTRICULAR DYSFUNCTION

Leung et al, Doherty et and Chia et al showed in their studies that left ventricular dysfunction occurs in about 4 – 71% of SLE patients.[12,76,77]

In this study, left ventricular dysfunction occurred in 10 patients , of whom 4 patients had systolic dysfunction and 6 patients had diastolic dysfunction. Overall 20% of the patients had left ventricular dysfunction, this finding also tally with the literature cited.

GLOBAL OR REGIONAL HYPOKINESIA

Doherty et al, Sturfelt et al showed that Global hypokinesia of myocardium as evidenced by echocardiography occurs in 8 - 12% of patients and regional hypokinesia in 7 – 16 % of the patients[75, 77]

In this study Global hypokinesia was seen in 4 patients accounting for 8% and Regional hypokinesia was seen in 6 patints accounting for 12%. this finding also coincides with the literature.

CONCLUSION

In our study 72% of the patients had cardiac manifestations.

The commonest cardiac manifestation is Pericarditis/ Pericardial effusion (40%).

Valvular disease is the second most common cardiac manifestation next to Pericarditis, (34%) with Mitral regurgitation/Mitral valve prolapse being the most common valvular disease, next common being Aortic regurgitation and the least common valvular abnormality is Tricuspid regurgitation.

In this study Systemic hypertension is found in 58% of the patients and pulmonary hypertension in 6% of the patients.

Left ventricular dysfunction is found in 20% of the patients, with systolic dysfunction being 8% and the diastolic dysfunction being 12%.

The commonest Arrhythmia found in this study is Sinus Tachycardia (46%) and the conduction disturbances noted are Right Bundle Branch Block(6%), Left Anterior Hemi Block(4%) and AV block (2%).

The least common cardiac finding is Libman – Sacks endocarditis, which is found in one case only (2%).

Almost all the cardiac findings are in par with what is seen in literature except for higher prevalence of systemic hypertension and lower prevalence of Libman – Sacks endocarditis.

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PROFORMA

A STUDY ON CARDIOVASCULAR MANIFESTATIONS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS.

Name Age Sex

IP/OP number Address

HISTORY:

SYMPTOMS

Chest pain

Dyspnea

Palpitations

Photosensitivity

Oral ulcers

Hematuria

Seizures

Hallucinations

Abnormal behaviour

Fetal wastage

Raynaud's phenomenon

ON EXAMINATION:

SIGNS

Pericardial rub

Pleural rub

Oral ulcers

Arthritis

Malar rash / Discoid rash

Petechiae

Purpura

Lymphadenopathy

Elevated JVP

Pulse rate:

Blood pressure:

Cardio vascular system:

Respiratory system:

Abdomen:

Central nervous system:

INVESTIGATIONS:

Urine albumin

Urine sugar

Urine deposits

24 hours urinary protein

Renal biopsy

Complete blood count

ESR

Antinuclear antibody

ds DNA

Antiphospholipid antibody

X ray chest PA view

ECG

ECHO

Sl no	Name of the patient	Age	sex	Ip/op number	Clinical symptoms							signs								Pul Se/mln	BP mm /hg	24 Hrs Urine Protein (mg)	ANA	ESR mm / hr	ECG								ECHO										
					Chest Pain	Dysponea	Palpitation	SYncope	Rayphnomen	PhotoSens	Seizurees	Malar Rash	Arthritis	Jvp	A2	P2	Basal crac	Pericarrub	S3						ST ↓	PR ↓	LVH strain	Sinus tacy	T ↓	LVC	RBBB	LAHB	Pericadef	MVPS	MR	AR	TR	PHT	Vegetation	Sys Dysfun	Dia dysfun	Reg Hypkn	Glob Hypkn
1	Kali ammal	50	F	049807		+			+			+		+		+				88	130/80	1000	+	80										+	+	+	+					
2	parameshwari	22	F	80609	+					+										72	160/90	790	_	60			+													+		
3	Kalai arasi	27	F	1044755		+			+			+								86	110/70	820	+	80																		
4	Muthu	33	M	1100827	+	+				+			+					+		112	116/70	650	+	68	+			+					+									
5	Saravana	38	F	1042001			+					+								84	130/80	780	+	54									+		+							
6	vimala	48	F	141707	+	+			+	+	+	+			+					103	160/100	960	+	50			+	+	+													
7	Geetha	33	F	158409	+					+				+				+	+	109	120/60	1030	+	86	+								+	+	+							
8	priya	27	F	1062573	+				+	+										61	160/90	980	+	34			+		+											+		
9	usha	25	F	1064079	+					+					+					110	150/100	590	+	50				+					+	+								
10	Amsavalli	35	F	1064738	+							+			+					70	160/100	820	+	38																		
11	Karthick	13	M	1091477	+	+		+		+		+					+		+	138	90/60	945	+	84	+			+	+				+						+			+
12	Saranya	17	F	1063104						+										80	140/90	860	+	56																		
13	Thangam	50	F	1059231	+		+		+											123	110/70	1250	+	76				+		+			+									
14	Sathya	21	F	1063085		+						+			+					106	150/90	710	+	39				+						+								
15	Angar begam	32	F	1056749		+			+			+		+			+			118	106/70	880	+	70				+					+	+	+			+	+		+	

Sl no	Name of the patient	Age	sex	Ip/op number	Clinical symptoms							signs								Pulse/mln	BP mm/hg	24 Hrs Urine Protein (mg)	ANA	ESR mm/hr	ECG							ECHO										
					Chest Pain	Dysponea	Palpitation	SYncope	Ray phnomen	Photo Sens	Seizures	Malar Rash	Arthritis	Jvp	A2	P2	Basal crac	Pericar rub	S3						ST ↓	PR ↓	LVH strain	Sinus Tacy	T ↓	LVC	RBBB	LAHB	Pericadef	MVPS	MR	AR	TR	PHT	Vegetation	Sys Dysfun	Dia dysfun	Reg Hypkn	Glob Hypkn
16	Dhana lakshmi	35	F	1056386	+					+		+						+		110	120/70	650	+	64				+	+		+	+	+	+	+								
17	udhayanandini	19	F	1653398	+				+				+							89	150/90	580	+	56			+															
18	Rekha	27	F	1052778	+			+		+		+								82	146/90	980	+	48			+		+										+	+	
19	Shanthi	40	F	1048897		+	+		+	+		+								105	154/90	1700	+	120				+	+				+		+	+					
20	Udhaya sheela	18	F	1053409		+					+				+					72	150/90	720	+	50																	
21	Lakshmi	27	F	1004588	+				+	+										109	110/50	1560	_	80				+	+			+	+	+	+	+						+
22	Sounder nayagi	40	F	1045324	+					+		+								94	110/70	2100	+	70																	
23	Jaya kodi	37	F	104800	+				+			+								105	160/100	790	+	46			+	+													
24	Uma shankari	18	F	1037921	+					+										109	120/60	950	+	64		+		+				+	+	+						+	
25	Malliga	37	F	1042497	+				+	+		+			+					105	160/100	750	+	45			+	+	+												
26	Fathima beevi	39	F	1033056		+	+					+								117	150/90	3100	+	82				+				+	+							+	
27	Ishwarya	26	F	1071829	+													+		107	150/94	1990	+	76				+	+				+	+							
28	Divya	29	F	1080673	+				+						+					68	174/110	690	_	50																	
29	Gomathi	22	F	1088786			+			+		+								92	100/70	2700	+	50					+		+										
30	Sentamil selvi	36	F	1081527		+			+											102	120/70	800	+	86		+		+			+		+								

no	Name of the patient	Age	sex	Ip/op number	Clinical symptoms							signs								Pulse/mln	BP mm /hg	24 Hrs Urine Protein (mg)	ANA	ESR mm/hr	ECG								ECHO										
					Chest Pain	Dyspnoea	Palpitation	SYncope	Ray phnomen	Photo Sens	Seizures	Malar Rash	Arthritis	Jvp	A2	P2	Basal crac	Peri car rub	S3					ST ↓	PR ↓	LVH strain	Sinus Tacy	T ↓	LVC	RBBB	LAHB	Pericad ef	MVPS	MR	AR	TR	PHT	Vegetation	Sys Dysfun	Dia dysfun	Reg Hypkn	Glob Hypkn
31	Sivasamy	60	M	1092577	+										+					96	150/100	655	+	90																	
32	Vijaya kumarai	30	F	1091788	+					+		+	+							82	170/90	975	+	36			+		+										+		
33	Amutha	32	F	1091771	+					+		+						+		104	100/70	2400	+	76				+	+				+	+	+						
34	Sudha	32	F	9857/10	+		+		+	+										112	120/80	711	+	60	+			+													
35	Jaya lakshmi	45	F	113106	+		+						+							89	160/100	1200	+	80			+		+										+		
36	Kanaga	21	F	1072663						+										88	154/90	615	+	58																	
37	Abinaya	16	F	129210	+															104	110/70	4900	+	100				+						+	+	+				+	
38	Kanaga valli	32	F	126110	+						+	+			+					113	144/100	590	+	58				+	+						+	+					
39	Mumtaj	18	F	0028/10	+	+		+		+		+		+		+	+		+	106	150/64	600	+	110			+	+						+	+	+	+	+	+		+			+
40	Jothi	38	F	1051412	+			+				+								73	148/94	1700	+	68																	
41	Ravi chandran	33	M	1006238	+															82	160/100	960	+	50																	
42	Panjavarnam	43	F	1061491	+					+										85	120/70	850	+	29																	
43	Gandhimathi	25	F	1102511	+	+								+		+	+	+	+	110	130/90	710	+	40		+		+						+	+	+		+	+		+			+
44	Latha	24	F	803/09		+	+								+					80	150/100	2010	+	44																	
45	Sabapathy	35	M	1013235	+															109	100/80	1030	+	60				+	+	+			+								

Sl no	Name of the patient	Age	sex	Ip/op number	Clinical symptoms							signs									Pulse/mln	BP mm/hg	24 Hrs Urine Protein (mg)	ANA	ESR mm/hr	ECG							ECHO											
					Chest Pain	Dyspnoea	Palpitation	SYncope	Ray phnomen	Photo Sens	Seizures	Malar Rash	Arthritis	Jvp	A2	P2	Basal crac	Pericar rub	S3						ST↓	PR↓	LVH strain	Sinus Taccy	T↓	LVC	RBBB	LAHB	Pericad ef	MVPS	MR	AR	TR	PHT	Vegetation	Sys Dysfun	Dia dysfun	Reg Hypkn	Glob Hypkn
46	Rani	32	F	1093733	+																94	146/100	510	+	46																	
47	Kannagi	28	F	1083764	+		+			+			+					+			92	160/90	970	+	64					+			+								
48	Menaka	24	F	1077432	+				+			+									78	130/80	860	+	54						+		+							+	
49	Bavya	17	F	1012654	+					+		+									88	144/90	560	_	46																
50	Susila	45	F	1289/10	+																88	150/100	690	+	80			+		+										+		

Ray phenomen - Raynaud's Phenomenon

Photo sens - Photosensitivity

JVP – Jugular Venous Pulse

Basal crac – Basal crackles

A2 - Aortic component of second heart sound

P2 - Pulmonary component of second heart sound

S3 – Third Heart Sound

Pericard Ef - Pericardial Effusion

Pericar rub – Pericardial rub

BP – Blood Pressure

ANA – Anti Nuclear Antibody

ESR – Erythrocyte Sedimentation Rate

ST↓ - ST Segment Depression

PR↓ - PR Segment Depression

T↓ - T Wave Inversion

MVPS – Mitral Valve Prolapse Syndrome

LVH – Left Ventricular Hypertrophy

Sinus Taccy – Sinus Taccycardia

LVC – Low Voltage Complexes

RBBB – Right Bundle Branch Block

LAHB – Left Anterior Hemi Block

MR – Mitral Regurgitation

AR – Aortic Regurgitation

TR – Tricuspid Regurgitation

PHT - pulmonary Hypertension

Sys dysfun – systolic Dysfunction

Dia dysfun – Diastolic Dysfuncyion

Reg Hyp Kn – Regional Hypokinesia

Glob Hyp Kn – Global Hypokinesia

ECG – Electrocardiogram

ECHO – Echo

IP/OP no – Inpatient /Outpatient number