TRANSESOPHAGEAL ECHOCARDIOGRAPHY IN THE DETECTION OF POTENTIAL CARDIAC SOURCE OF EMBOLISM IN YOUNG STROKE PATIENTS

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CERTIFICATE

Certified that this dissertation entitled "**TRANSESOPHAGEAL ECHOCARDIOGRAPHY IN THE DETECTION OF POTENTIAL CARDIAC SOURCE OF EMBOLISM IN YOUNG STROKE PATIENTS"** is a bonafide work done by **Dr.B.KANNAN,** Post Graduate Student of Internal Medicine, Institute of Internal Medicine, Madras Medical College, Chennai - 600 003, during the academic year 2003- 2006.

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DECLARATION

I solemnly declare that the dissertation entitled **"TRANSESOPHAGEAL ECHOCARDIOGRAPHY IN THE DETECTION OF POTENTIAL CARDIAC SOURCE OF EMBOLISM IN YOUNG STROKE PATIENTS "** is done by me at Madras Medical College and Hospital, during 2004-2006 under the guidance and supervision of **Prof.V.K.RAJAMANI, M.D.** This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfilment of requirements for the award of **M.D. DEGREE IN GENERAL MEDICINE (BRANCH I)**.

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CONTENTS

INTRODUCTION

Ischemic stroke is one of the major causes of morbidity and mortality in middle and late years of life. The incidence of stroke increases with age, this disability affects many people in their golden years. Stroke is the 3rd leading cause of death preceded only by cardiac disease and malignancy. Cerebrovascular accident is an abrupt onset of non-convulsive and focal necrologic defect.85% CVA caused by ischemia, out of which 20% of is constituted by cardio embolic stroke[5].

Cardio embolic stroke is defined as non-lacunar stroke with presence of potential cardiac source in the absence of cerebrovascular diseases. It has an unfavourable outcome since it produces larger and more disabling stroke than other subtypes. Cardiac embolic stroke is largely preventable, rendering measures of primary prevention valuable. Once the stroke has occurred the likelihood of recurrence is high, thus secondary prevention is also equally important[32].

The availability of new diagnostic techniques especially transesophageal echocardiography (TEE) has allowed clinicians to identify cardiac source of embolism. TEE is a new application of endocardiography that allows high resolution imaging of atria, atrioventricular valves and aorta by inserting an ultrasonic transducer in the esophagus[10].

Recent studies suggest that transesophageal echocardiography may detect a potential source in upto 65%[5] of patients with cerebral ischemic events

or systemic arterial embolism. This study was undertaken to compare the diagnostic yield of transesophageal echocardiography with transthoracic echocardiography in the detection of a cardiac source of embolism in patient with cerebral ischemic events under the age 45 yrs.

AIM OF STUDY

1) To compare the diagnostic yield of transesophageal echocardiography with transthoracic echocardiography in the detection of cardiac source of embolism in patients with cerebral ischemic events.

2) To document intracardiac lesions which could have been the source of embolism for ischemic stroke.

3) To evaluate the presence of clots in cardiac chambers.

4) To find out incidence of patent foramen ovale (PFO) in this study population.

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REVIEW OF LITERATURE

Stroke or cerebrovascular accident[25] is defined as abrupt onset of a neurologic deficit that is attributable to focal vascular cause. Thus the definition of stroke is clinical and laboratory studies including brain imaging are used to support the diagnosis.

Cerebral ischemia is caused by a reduction in blood flow that lasts longer than seconds. Neurological symptoms manifest within seconds because neurons lack glycogen, so energy failure is rapid. When blood flow recovers rapidly, brain tissue recovers fully and patients' symptoms are only transient. This is called transient ischemic attack(TIA).

In TIA[25], typically the neurological signs and symptoms last for 5 to 15min but, by definition must last <24 hrs.

Stroke has occurred if neurological signs and symptoms last >24 hrs. i.e. cessation of blood flow for more than few minutes leads to infarction or death of brain tissue.

Generalized reduction in cerebral blood flow due to systemic hypotension usually produces syncope. If low cerebral blood flow persists for long duration, then infarction in border zones between major cerebral artery distribution develops.

In more severe instances, global hypoxia-ischemia causes wide spread brain injury, the constellation of cognitive sequelae that ensues is hypoxic-ischemic encephalopathy.

Stroke divided into ischemic and haemorrhagic out of which ischemic constitutes 85%; Haemorrhage form 15%[25].

Atherothrombotic stroke-stroke produced by thrombosis of large intracranial vessel (0.5 to 3mm) in situ from atherosclerosis[25].

Lacunar stroke-thrombotic occlusion of small intracranial vessels (30 to 100 μm) which supply small volume brain, because they are end arteries. They result in stroke called lacunar stroke. Low flow stroke or transient ischemic attack if cardiac output or systemic B.P. reduced below some threshold.

Cerebral embolism refers to an obstruction of blood vessel in brain by an embolism originating any where in the circulatory system. Cardiogenic embolism is recognized increasingly as an important cause of stroke. Approximately 20% of all ischemic stroke are cardioembolic[25].

Cardio embolic stroke

The frequency of cardio embolic stroke in patients younger than 45 year age in much higher ranging from 23% to 36%[37].

New diagnostic techniques have allowed clinicians to better characterize well established sources of embolism and to discover other potential etiologies of cardio embolic stroke.

Cardio embolic stroke largely preventable warranting efforts at primary prevention for major risk cardio embolic sources. Once stroke due to cardio embolism has occurred, the likelihood of recurrence is relatively high for most cardio embolic sources and consequently secondary prevention is also important.

Pathophysiology

No single mechanism is responsible for the development of cardiac emboli. The specific underlying cardiac diseases determines pathophysiology and natural history and hence each cardioembolic stroke must be considered separately.

Traditionally cardioembolic stroke have been regarded as severe and disabling. Undoubtedly large cortical infarction with hemorrhagic transformation are commonly of cardiac origin. These findings result from embolization of sufficiently large quantities of thrombus material or large vegetations, Constituting many of the cardio genic emboli.

Other cardiogenic emboli smaller; the associated hemispheric events may be transient or minor with small area of infarction or they may be retinal and present as transient monocular blindness or retinal infarction.

The cerebral circulation absorbs 10% to 15% of cardiac output, carotid artery blood flow accounts for approximately 90% of total cerebral blood flow. The most commonly site of lodgement of cardiac emboli are main trunk and branches of middle cerebral artery, only 7% lodged in anterior cerebral artery, embolism into anterior cerebral artery is rare[17].

About 10% of cerebral emboli enter the vertebrobasilar circulation where they lodge in main trunk or in one of the branches of posterior cerebral arteries. Once emboli have reached the cerebral circulation it causes ischemia, in contrast to thrombi, emboli are attached loosely to vascular walls and this commonly migrate distally. When this occurs reperfusion causes blood to leak into surrounding infarcted tissue. This explain the more frequent association of haemorrhagic infarction with cardiogenic embolism than with other causes of ischemic stroke, which occurs from 12 to 36 hr after embolization and often asymptomatic.

In the great majority of patients haemorrhagic transformation does not cause clinical worsening because the bleeding involves necrotic tissue.

Frequency

14-31% of ischemic stroke are cardio embolic depends on criteria and study design[32].

Consistent geographic variation is not evident and frequency is likely similar throughout if adjusted for mean population age.

Mortality/morbidity

In general cardioembolic strokes have a worst prognosis and produce larger and more disabling strokes than other ischemic stroke subtypes because emboli of large size from cardiac chambers.

Sex : Female to male ratio increase with age

Age : Bimodal higher in young less than 50 years and very old >75 yrs.

History

Although not sufficiently sensitive or specific to establish the diagnosis, several clinical features help to distinguish cardiogenic embolism from other mechanism.

- Decreased level of consciousness at onset of stroke
- Sudden onset of symptoms and signs that are maximal at onset.
- Rapid recovery from major hemispheric deficit due to reperfusion of brain with early lysis of the embolus.
- Onset of symptoms after a valsalva provoking activity (enhancing right to left shunt in patient with patent foramen ovale).
- Symptoms reflecting involvement of different vascular territories of brain.
- Certain neurologic syndromes suggest embolism often cardio embolism as their cause. They are

I. In the middle cerebral artery territory

a. Frontal opercular with facial weakness and severe aphasia or dysarthria.

b. The brachial or hand plegia syndrome which the arm or hand is paralysed with or without cortical sensory abnormalities.

c. Broca's or wernicke's aphasia alone

d. Left visual neglect-non dominant parietal lobe.

II. Posterior cerebral artery embolus-Sudden hemianopia

III. Anterior cerebral artery territory: Sudden weakness of foot and shoulder.

IV. Sudden sleepiness and inability to look up associated with bilateral ptosis suggest an embolism to the **top of basilar artery** specifically to the artery of percheron.

- Seizures at time of stroke occurs in 3 to 5% of infarctions most often associated embolic rather than thrombosis[25].
- Cardiogenic embolic (especially from chamber) do not often affect deep penetrating arteries as a lacunar syndrome, small cardiogenic emboli from valvular source can obstruct the small penetrating arteries causing subcortical lacunar infarcts.

Physical

1. Evidence of cardiac atrial dysrhythmias (eg, atrial fibrillation, sick sinus syndrome).

2. Presence of cardiac murmurs (eg, mitral stenosis, calcific aortic stenosis)

3. Signs of congestive heart failure (eg, after acute myocardial infarction, nonischemic cardiomyopathies).

4. Concomitant diseases (eg, systemic lupus erythematosus and Libman-Sachs endocarditis, neoplasia, marantic endocarditis).

5. Concomitant signs of systemic embolism

6. The probability of finding such signs in patients with suspected cardioembolic stroke is low (approximately 1%) for most cardioembolic sources.

7. The diagnosis of cardioembolic stroke is based on the triad[32] (1) identification of a potential cardioembolic source, (2) absence of other likely causes of stroke, and (3) supportive clinical features described above.

Causes

More than 20 specific cardiac disorders have been implicated in leading to brain embolism. Dividing cardiac sources of embolic into major-and minor-

risk categories is clinically useful (see below). Major-risk sources carry a relatively high risk of initial and recurrent stroke convincingly linked to a cardioembolic mechanism. When a major-risk cardioembolic source is present, efforts at primary prevention of stroke usually are indicated; stroke in patients with any of these causes is most often cardioembolic. Minor-risk sources are frequent in the general population, and the associated risk of initial and recurrent stroke with any of these conditions is either low or uncertain. When a minor-risk cardioembolic source is present in a patient with cerebral ischemia, the etiologic role must be viewed with skepticism and considered in the context of other diagnostic information.

Source of cardioembolic embolism include the following: Asterix (*) indicates a major-risk source; dagger (†) indicates emboli originating in the venous circulation or right heart that cause ischemic stroke via abnormal cardiac or pulmonary shunting around the pulmonary capillary bed.

Valvular disease

- Rheumatic mitral stenosis*
- Prosthetic valves*
- Calcific aortic stenosis
- Bicuspid aortic valves
- Mitral annulus calcification

- Nonbacterial thrombotic (marantic) endocarditis* associated with malignancies and prothrombic states
- Myxomatous mitral valvulopathy with prolapse
- Infective endocarditis*
- Inflammatory valvulitis (ie, Libman-Sacks endocarditis, Behcet disease, syphilis)
- Lambl excrescences and/or strands

Left ventricular thrombi

- Ischemic heart disease*
- Acute myocardial infarction*
- Left ventricular akinesis or aneurysm*
- Idiopathic hypertrophic subaortic stenosis
- Trauma (myocardial contusion)
- Ventricular non compaction
- Nonischemic cardiomyopathies* (eg, idiopathic dilating viral myocarditis associated, echinococcal, peripartum, amyloid-associated, hypereosinophilia syndrome-associated, rheumatic myocarditis-associated, sarcoidosis-related, neuromuscular disorder-associated, alcoholism-related, catecholamine-induced, Chagas disease-associated, doxorubicin-induced, mitoxantrone-related, crack cocaine-related, cardiac oxalosis-associated).

- Left ventricular thrombi associated with prothrombotic states*
- Antiphospholipid antibodies
- Diffuse intravascular coagulation
- Essential thrombocythemia and myeloproliferative diseases

Left atrial thrombi

- Arrhythmias
- Atrial fibrillation*
- Sick sinus syndrome/atrial asystole*
- Atrial flutter*
- Atrial septal aneurysms
- Chiari network

Cardiac tumours

- Atrial myxoma
- Cardiac sarcoma
- Endocardial fibroelastoma
- Metastatic disease

Paradoxical emboli†

- Atrial septal defects
- PFO
- Ventricular septal defects
- Pulmonary arteriovenous fistulas

Miscellaneous

- Postcardiac catheterization
- Post valvuloplasty
- Esophageal-atrial fistula

Atrial fibrillation: The leading cause of cardioembolic stroke, especially in elderly individuals, atrial fibrillation is percent in approximately 1% of the US population and in approximately 5% of those older than 70 years[32].

Formerly associated with rheumatic valvular disease, it now is related most frequently to hypertension and ischemic heart disease (ie, nonvalvular atrial fibrillation).

Both paroxysmal and chronic atrial fibrillation are associated with increased risk of stroke. Stasis secondary to decreased contractility of the left atrium leading to thrombus formation in its appendage is the postulated mechanism.

In a autopsy study of 642 patients with atrial fibrillation of various causes, a left atrial thrombus was found in 15.8% as opposed to 1.7% in 642 age of sex matched controls[1].

Left ventricular thrombus was encountered in 1% of patient with atrial fibrillation. Cerebral infarction occurred in 32.2% of the patients with atrial fibrillation and in only 11% of the controls[1].

The frequency cerebral infarction increased with duration of fibrillation the most common causes of atrial fibrillation were rheumatic and ischemic heart diseases. Presence of atrial fibrillation in acute stroke associated with worsened prognosis at 1 to 6 months[6].

Non valvular atrial fibrillation is a major cardioembolic stroke as well as of massive cerebral infarctions.

Transesophageal echocardiography is more sensitive than trans thoracic echocardiography for visualization of left atrium and its appendage[5].

Not all atrial fibrillation associated strokes are cardio embolic, in individual cases excluding other potential causes of stroke such as intrinsic cerebrovascular disease or aortic atheroma.

The annual rate of stroke in atrial fibrillation varies widely from 0.5-12% per year depending on prevalence and combination of risk factors; thus, risk stratification is the first necessary step in choosing the best preventive therapy. Several clinical risk stratification schemes have been proposed to identify atrial fibrillation at high, moderate, or low risk; this is crucial for selecting which patients would benefit most and least from anticoagulation. The CHADS2 (ie, CHF, hypertension, age >75 y, diabetes, stroke or TIA) classification scheme is the most validated and accurately stratifies stroke risk (Gage, 2001[13A]; Go, 2003[19]; Gage, 2004)[18].

Two randomized controlled trials have demonstrated that a strategy aimed at restoring (and maintaining) sinus rhythm neither improves the survival rate nor reduces the risk of stroke. In the atrial fibrillation follow-up

investigation of Rhythm Management (AFFIRM) study[28], 4060 patients aged 65 years or older whose atrial fibrillation was likely to be recurrent and who were at risk for stroke were randomized to a strategy of rhythm control (cardioversion to sinus rhythm, plus a drug(s) to maintain sinus rhythm) versus a strategy of rate control (in which no attempt was made to restore or maintain normal sinus rhythm). An insignificant trend toward increased mortality was noted in the rate control group and importantly, no evidence suggested that the rhythm control strategy protected patients from stroke.

The AFFIRM study (and similar findings from the smaller Rate Control Versus Electrical Cardioversion [Race][21] trial) has led to the development of consensus guidelines advocating a rate-control strategy for most patients with atrial fibrillation.

Adjusted-dose warfarin (international normalized ratio [INR] 2-3) is associated with a 60% reduction in stroke incidence, while the efficacy of aspirin is modest (20%). Low-dose warfarin (INR<1.5), either alone or combined with aspirin, is not effective, highlighting the marginal benefit of warfarin when anticoagulation is not carefully regulated. Incidence of intracerebral hemorrhage, the most dreaded complication of warfarin therapy, is estimated to be 0.5% per year among elderly patients with atrial fibrillation and is sensitive to blood pressure control. When warfarin is given to elderly patients with atrial fibrillation, hypertension must be managed aggressively (Hart, 2005)[22].

Recommendations for primary and secondary prevention based on risk factor stratification are presented in (Table 3).

In the setting of acute stroke secondary to atrial fibrillation, anticoagulation with heparin has not demonstrated more benefit than early treatment with aspirin. Initiate aspirin early, followed by warfarin as soon as the patient is medically stable; discontinue aspirin after therapeutic anticoagulation is achieved (Hart, 2003).

In short, warfarin has demonstrated high efficacy in stroke prevention in patients with this common arrhythmia. Disadvantages include the increased risk of hemorrhagic complications and the need for close INR monitoring in these patients; thus, consider patient preferences along with risk stratification and absolute risk reduction offered by this therapy. Alternative approaches (eg, surgical ablation of atrial appendage) are the subjects of ongoing clinical investigation.

Ximelagatran, a new oral thrombin inhibitor, is comparable to adjusted-dose warfarin for stroke prevention in patients with atrial fibrillation. The Stroke Prevention Using an Oral Direct Thrombin Inhibitor in Patients with Atrial Fibrillation (SPORTIF)[29] III and V clinical trials recently demonstrated that treatment of atrial fibrillation with ximelagatran twice daily is equivalent to adjusted-dose warfarin when considering stroke and major hemorrhage, but INR monitoring and dosage adjustments are unnecessary. However, potential liver toxicity led to disapproval by the US Food and Drug Administration (FDA) in late 2004.

Stroke rates by CHADS2 score*

CHADS2 Score	Risk	Stroke Rate Per Year
0	Low	1%
1	Low	1.5%
2	Moderate	2.5%
3	High	5%
≥	Very high	>7%

*Score determined by: CHF, hypertension, age>75, and/or diabetes=1 point; stroke or TIA=2 points.

Table : 3

Prevention of stroke in nonvalvular atrial fibrillation-recommendations

Prevention	Risk group	Recommended prevention	Range of Management
Primary prevention*	Lone AF†, <60 y	None	Aspirin 325 mg/d‡
	Low risk	Aspirin 325 mg/d‡	Warfarin, INR 1.6-3.0,
	Moderate risk	Warfarin, INR 1.6-3.0, or aspirin 325 mg/d	--
	High risk, >75 y	Warfarin, INR 2.0-3.0	Aspirin if warfarin contraindicated
	High risk >75 y	Warfarin, INR 1.6-2.5	Aspirin if warfarin contraindicated
Secondary prevention		Warfarin, INR 2.0-3.0	Aspirin if warfarin contraindicated

*See table 2 for current CHADS2 stratification schemes.

†AF indicates atrial fibrillation.

‡ Lower doses of aspirin are probably equally efficacious; the value of aspirin combined with clopidogrel 75 mg/d is being tested in a large randomized trial.

Sick sinus syndrome (SSS)

Any form of sinus node depression such as marked sins bradycardia (<50 beats/min), sinus arrest or sino atrial block.

The SSS occurs throughout life, but is more common in older patients.

It may be associated with IHD, cardiomyopathy and neuromuscular disease but is frequently idiopathic or degenerative in origin.

The clinical manifestation are mainly those provided by transient global ischemia (Light headedness and syncope). However patients with SSS are at high risk for stroke, which is usually massive in presumably cardioembolic in origin.

The only effective treatment for SSS is pacemaker implantation, but the stroke rate is still high after pacing.

Valvular heart disease

Rheumatic heart disease (RHD)

Clinical embolic events occur in about 20% patients with RHD[9].

Embolism usually complicates mitral stenosis or mixed stenosis regurgitation. Whereas pure mitral regurgitation or aortic disease less frequently results in embolism[9,11].

Mitral stenosis

Mitral stenosis is most often of rheumatic origin, left atrial thrombi form in a large number of affected patients, particularly in the presence of atrial fibrillation or cardiac failure.

Left atrial thrombus is present in 15% to 17% of patients at autopsy, regardless of whether or not there has been a H/o of embolism[9].

Thrombi may develop in patients with only Mild mitral stenosis, explaining why embolism may be a presenting symptoms of mitral stenosis.

In patients with mitral stenosis the annual incidence of all symptomatic emboli is about 4% more than half are cerebral but many asymptomatic systemic emboli and occasional asymptomatic cerebral embolic are found at postmortem[9].

Recurrent embolism is common (30% to 75%) usually within 6 to 12 months of sentinal event and the presence of atrial fibrillation increases the risk embolism four fold. The risk of embolism also increases with increasing duration of mitral stenosis[39].

Spontaneous echo contrast is a dynamic smoke like signal that is detected by TEE in patients with stasis of blood in left atrium 42 patients with mitral stenosis or atrial fibrillation spontaneous echo contrast compared patient without spontaneous echo contrast, showed spontaneous echo contrast is highly associated with previous stroke or peripheral embolism in patient with atrial fibrillation or mitral stenosis[7].

Embolisation of left atrial thrombi account for >45% of cardiogenic emboli, left atrial thrombi are most often associated with atrial fibrillation or rheumatic mitral stenosis.

The sensitivity of transthoracic echo cardiography for the detection of left atrial thrombi is only 39-63%[33] in contrast TEE offers detailed visualization of both body of left atrium and left atrial appendage[5].

Careful discrimination of thrombi from left atrial pectinate muscles, as well as familiarity of multi lobed appearance of the normal left atrial appendage can be better appreciated with multiplane TEE[5].

Long term anticoagulation recommended for prophylaxis in patients with rheumatic mitral stenosis and coexistent atrial fibrillation, as combination of both carries a 17 fold increase in stroke risk compared with matched controls[38].

Then embolic and recurrent emboli rates are notably decreased with long term anticoagulation although long term therapy is not totally protective.

Mitral regurgitation

Mitral regurgitation most commonly results from mitral valve prolapse or papillary muscle dysfunction association with ischemic or rheumatic heart disease.

When severe the mitral regurgitation produces an ulcerated "Jet lesion" on atrial endocardial surface, left atrial thrombus may form there, this occurs in some what less than 10% of patients and almost invariably in the presence of atrial fibrillation[36].

Thrombo embolism is a recognised complication of mitral regurgitation. The incidence is low even though atrial fibrillation is a common accompaniment particularly late in course.

Aortic stenosis

Isolated aortic stenosis is mainly caused by calcification of valve cusps, a process observed predominately in elderly people. Most patients seen with cerebral embolism have had multivalvular rheumatic diseases or coexistent infective endocarditis[27].

Introduction of echo, doppler and catheterization it has become evident that calcific aortic stenosis may be responsible for more cerebrovascular events than recognized.

Calcific micro emboli can be detected in the retinal artery is asymptomatic patients possibly reflecting the fact that most cerebral emboli are asymptomatic. Clinical embolism often follows invasive cardiac procedure (catheterization).

Because of the calcific nature of embolic anticoagulation is not recommended.

Mitral annular calcification

This is associated with advancing age, hypertension and atherosclerosis and it is rarely embolic source[15].

Mitral valve prolapse

Mitral valve prolapse a common finding in young, carries a benign course for most individuals and is an unusual cause of stroke among those harboring mitral valve prolapse. It should the considered as the underlying cause for stroke only, if extensive search for other causes has been negative.

Prophylactic treatment is unwarranted in individuals with asymptomatic mitral valve prolapse in patients where cerebral ischemia is attributed to mitral valve prolapse aspirin therapy is recommended on empirical grounds and only in treatment failures ,when further events occur, should anticoagulants be used.

Idiopathic hyper trophic subaortic stenosis (IHSS)

Idiopathic hyper trophic subaortic stenosis is felt to carry a low risk of embolism. Atrial fibrillation is often associated in there into do have presumed cardioembolic source[16].

Prosthetic valves

Thromboembolism remains a major cause of morbidity and mortality complicating prosthetic valve implantation. The majority of thrombo embolic events are cerebrovascular. There are many kinds of mechanical valves all of which require life long anticoagulation therapy to prevent thrombo embolism.

Even such treatment, patients with mechanical valves experience further embolic events at a rate comparable to non anticoagulated patients with bioprosthetic valves.

The rate of embolism in patients with mechanical valve averages 3% to 4% /yr. In aortic position the rate estimated to be lower averaging 1.2% to 2.2% / yr[13].

When bioprosthetic valve is inserted long term anticoagulation is not recommended when other risk factors are absent.

Permanent anticoagulation (INR 2.5-3.5) is mandatory for prosthetic valves .For bioprosthetic valves aspirin usually is recommended unless the patient has atrial fibrillation or evidence of atrial stasis.

Prosthetic valve endocarditis is another complication and a potential source of cerebral embolism, incidence 2.4%/yr. In patient with prosthetic valves, occurrence of neurologic complications is higher with mechanical valve mitral in position[26].

Evaluation of mechanical and bioprosthetic valves for valvular function and exclusion of thrombi or vegetation is best performed by TEE.

Patient with mechanical prosthesis who present with evidence of systemic embolisation are generally assumed to have prosthetic valve thrombi especially when INR is low.

Acute myocardial infarction

The incidence of stroke often AMI is approximately 2% in the first 3 months. Acute myocardial infarction with mural thrombus on TTE have been recognized as predictive of stroke. Anticoagulation INR 2-3 is recommended in these patients in the 1st 3 months, while antiplatelet therapy is advocated long term.

The presence of congestive cardiac failure after Acute myocardial infarction usually dictates treatment with warfarin indefinitely. Low cardiac output failure (EF<30%) is also considered a high risk situation, as is the presence of a left ventricular aneurysm on echo.

Anticoagulation therapy should be discontinued after 3 to 6 months over if left ventricular thrombi persists. Routine anticoagulation has not been recommended if left ventricular thrombi are detected in aneurysmal sac at a time remote from Acute myocardial infarction[14].

Instead long term anticoagulation has been recommended only in patients with cerebral insults attributed to persistent left ventricular thrombi. The recommended treatment policy for patients with acute transmural myocardial infarction includes use of high dose of heparin, even if thrombolytic therapy is given. Echo should be performed 5 to 7 days later to detect left ventricular thrombi if no thrombus or wall motion abnormalities, heparin should be discontinued.

The line of TTE with higher frequency transducers (5.0 MHz) really identifies or excludes left ventricular thrombi in most patients, among patients with poor apical windows or those whom TTE data are equivocal, TEE is a certainly reasonable alternative for identifying these thrombi[5].

Dilated cardiomyopathies

Arrhythmias are common, either as nonsustained ventricular tachycardia or chronic atrial fibrillation, which develop 20 to 30% of patients. Thus arrhythmias and high prevalence of left ventricular thrombi that are found in dilated cardiomyopathies are believed to be the underlying cause for stroke and other embolic complication in these patients.

Echo detects the presence of thrombi in 11% to 58% of patients with idiopathic dilated cardiomyopathy, yet it is not helpful for detection of at high risk for stroke as there is no correlation has been found between the detection of thrombi and presence of emboli.

Infective endocarditis

Of patients with infective endocarditis, 20% experience an embolic stroke, staphylococcus aureus is the agent producing highest stroke rate, mitral valve endocarditis is the most common source of emboli. Antibiotic therapy reduces embolic potential when administered in the acute phase. Anticoagulation is contraindicated because of unacceptable rates of hemorrhagic stroke.

In patients prosthetic valve endocarditis the risk of thromboembolism is greater than the risk of ICH, this anticoagulation usually recommended if no evidence of haemorrhage is found on CT scanning 24-48 hrs after the stroke, consensus is to start anticoagulation 7 days after the stroke-the role of antiplatelet therapy has not been established.

Non bacterial thrombotic endocarditis

Associated with variety of malignancies like carcinoma lung, pancreas and prostate non bacterial thrombotic endocarditis also has been reported in patients with severe diseases such as septicaemia and extensive burns. Mitral and aortic valves are affected most commonly and embolic stroke is frequent, it is mainly found in normal cardiac valves.

A prothrombotic state has been postulated as the precursor of emboli development. The vegetations in NBTE are usually small and are composed of platelet and fibrin deposits.

Treatment is directed toward control of underlying disease and heparin (I-V in Acute stage, subcutaneous in the O-P setting) is advocated for stroke prevention. Warfarin failed to show any benefit.

Cardiac myxoma

Most common primary heart tumour . very rare cause of stroke.

The tumour is usually benign and originates in left atrium in 75% cases, primarily diagnosed in young and middle aged adults. It is usually located in the fossa ovalis. It may present with either constitutional, cardiac obstructive or embolic symptoms alone or in combination. Embolic manifestation occur in 20 to 45% of patients sometimes as the first symptoms. Although emboli may lodge in any vascular system CNS is involved in upto 50% cases. Embolic material consists of mainly myxomatous tissue.

Neurologic manifestation, consist mainly of acute focal neurological deficit that may be the initial presentation of disease. Most can be detected by TTE, rarely it can be detected only by TEE especially small and multiple as in familial myxoma syndrome. Surgical excision is the treatment of choice.

Paradoxical embolism

The name implies that an embolus originating in the venous system finds its way to lodge on the arterial tree and implies the existence of right to

left shunts possible shunts incriminated in pathophysiology of paradoxical embolism include patent foramen ovale, atrial septal defect and pulmonary A-V fistula.

PFO (patent foramen ovale)

Hemodynamically insignificant interatrial communication is present in over 25% of the adult population.

Because stroke occurs more frequently in older population with only 3% of cerebral infarction occurring in patients <40 yrs of age, The no. of stroke patient with PFO >40 yrs of age is much larger than in the younger patients. Several studies reported the association of PFO with cryptogenic stroke in older population. However this has not been in seen in all studies.

Therefore, although the association between cryptogenic stroke and PFO is established among the younger population, it is not clearly established in the older population.

Webster et al.[10A] performed contrast echocardiography in 40 patients with stroke or TIA all aged under 40 yrs, and 40 age and sex matched controls, A PFO was found in 50% of patients with cryptogenic stroke. Transthoracic echocardiography and transesophageal echocardiography with saline contrast are widely used to detect PFO.

PFO is judged to be present if any microbubble in seen in left side chambers with in 3 cardiac cycles from the maximum right atrial Opacification after injection of agitated saline is prepared by mixing 0.5 to 1 ml of air with 10

ml of NS through two syringes the peripheral I-V line[34]. Injection is performed with and without valsalva maneuver, cough during injection increase the sensitivity for detecting PFO over that achieved by valsalva maneuver.

TEE contrast study is the most sensitive diagnostic test available for detecting a PFO followed by TCD and transthoracic contrast studies.

In recent PFO in cryptogenic stroke study (PICSS) it also has been shown that large PFO's were significantly more prevalent among Cryptogenic stroke patients compared with those with known cause of stroke. Additionally stroke patients with larger PFO have brain imaging finding suggestive of an embolic phenomenon.

Deep vein thrombosis (DVT)

Stolleberger et al[35]. study demonstrated that patients with PFO who have paradoxical embolization may harbor the source of embolism in lower extremities. The frequency of DVT in cryptogenic stroke patients with PFO is not clearly defined although the most recent series indicates that is increased.

Atrial septal aneurysm (ASA)

ASA is a redundancy of interatrial septum detected most commonly by TTE or TEE studies. It is detected significantly more frequently using TEE study compared to TTE study. On TTE study defined as 15mm protrusion or excursion of interatrial septum with base of 15mm, in TEE it is >10mm.

The prevalence of ASA in patients with cerebral systemic embolic events is high and reaches 73%. It is well known that ASA is associated with PFO. It can be seen approximately 60% patients with ASA also have PFO.

When ASA associated with PFO others source of embolism anticoagulation usually recommended. Management is controversial because of lack of diagnostic certainty and absence of randomised evidence. Antiplatelet therapy is often advocated initially and warfarin is recommended empirically. If stroke recurs during antiplatelet therapy or if prothrombic diathesis predisposing to recurrent DVT.

SURGICAL CLOSURE OF PFO

The no. of patient in each one of the series is small for both percutaneous and surgical closure. Also the selection criteria vary among studies patient included them the young. Consequently the efficacy of percutaneous or surgical PFO closure remains undefined and is best reserved for young patients not able to undergo medical therapy.

MATERIALS AND METHODS

Stroke patients admitted in medical wards Govt. General Hospital during the period of June 2005 to March 2006, were selected for this study.

INCLUSION CRITERIA

1. Patients with or without cardiac disease below the age 45 yrs..

2. Patient without any known risk factors for thrombotic stroke.

Exclusion criteria

1) Age >45 yrs.

2) Patient with known risk factors for Atherothrombotic stroke like diabetes mellitus, systemic hypertension, smoking.

3) CT suggestive of haemorrhage.

Only inpatients were included in series to ensure that investigation, treatment and follow up of cases are done properly and efficiently

Detailed history was taken from patients who where conscious and not aphasic. In unconscious patients, history was taken from a reliable informant.

A thorough cardiovascular and neurological examination were carried out. After relevant investigation patients with hypertension, diabetes mellitus hyperlipidemia were excluded from the study.

METHODOLOGY

Patients with stroke less than 45 yrs were submitted to CT scan. If scan showed infarction, haemorrhagic transformation or multiple infarcts they were included in the study, pure haemorrhagic stroke were not included. Both transthoracic echocardiography & transesophageal echocardiography were done for all patients, on the same day by cardiologist who has adequate training. Standard parasternal and apical views were obtained using Aloka ultrasonic 2000 interfaced with 5MHZ transducer. Intra venous saline contrast was not given during transthoracic echocardiography.

Transesophageal colour doppler echocardiography was performed after obtaining informed Consent, by using multiplane transesophageal echocardiography 5MHZ transducer interfaced with Aloka 2000.

A complete multiplane transesophageal echocardiography done in all patients, 14 patients were given saline contrast during transesophageal echocardiography.

The following echocardiographic abnormalities were considered a possible cardiac source of embolism. Left atrial or left ventricular thrombi, left atrial spontaneous echocontrast[12], mitral stenosis, myxomatous degeneration of mitral valve with prolapse[3], severe mitral annular calcification, atrial septal defect, patent foramen ovale, atrial septal aneurysm, left ventricular aneurysm or apical hypokinesia, aortic or mitral vegetation, atrial myxoma, moderate and severe globular left ventricular hypokinesia and prosthetic heart valve.

STATISTICAL ANALYSIS

Qualitative data was expressed in frequencies with their percentage.

Quantitative data was expressed in mean standard deviation (SD).

Qualitative data was analysed (Sex, TTE, TEE) to compare the difference between those with clinical evidence of heart disease and those with no clinical evidence of heart disease by Pearson Chi-square test and Yates corrected chi-square test. Then odds ratio was given with 95% confidence interval.

Quantitative data (age) was analysed using student t-test to compare the significant difference between those with clinical evidence of heart disease and those with no clinical evidence of heart disease.

OBSERVATION AND RESULTS

35 patients with ischemic stroke below the age of 45 yrs were assessed clinically and biochemically . Three of these patients had diabetes, 2 could not swallow and were not included in the study. A total of 30 patients were included in the study

SEX DISTRIBUTION

Sex	No. of patients	Percentage
Male	17	56%
Female	13	44%

AGE DISTRIBUTION

Age	Male	Female	Total	Percentage
12-20	1	2	3	10%
21-30	2	3	5	17%
31-40	12	5	17	56%
41-45	2	3	5	17%

Mean age with maximum incidence is 31-40 (56%); minimum incidence was in the younger age group 12-20 (10%).

CLINICAL FEATURES

Based upon side of involvement and severity of weakness

	Right	Left
Hemiplegia	8	9
Hemiparesis	6	6
Posterior circulation	1	-

Aphasia was found in 12 (40%) cases. All of them had right hemiplegia.

These 30 patients were divided into two groups based on presence or absence of clinical evidence of heart disease.

GROUP I - 16 cases

PATIENTS WITH CLINICAL EVIDENCE OF HEART DISEASE

Age	Male	Female	Total	Percentage
<20	-	2	2	12%
21-30	1	2	3	19%
31-40	3	5	8	50%
41-45	-	3	3	19%

There is female predominance in Group I since rheumatic mitral stenosis is more common in females.

Both the patients less than 20 yrs had prosthetic valves in mitral position.

Group II (14 cases)

PATIENTS WITH NO CLINICAL EVIDENCE OF HEART DISEASE

Age	Male	Female	Total	Percentage
<20	1	-	1	8%
21-30	1	1	2	14%
31-40	9	-	9	64%
41-45	2	-	2	14%

Here there is a slight male preponderance and the age group affected maximum is 31-40.

STATISTICAL ANLAYSIS OF AGE IN GROUP I & GROUP II

	N	Mean age	SD	t-test
With clinical evidence of heart disease	16	32.87	8.53	E = 0.57 P = 0.58 Not significant
With no clinical evidence of heart disease	14	34.50	6.77	

In both Group I and Group II mean age was similar ,which is not statistically significant.

STATISTICAL ANALYSIS OF SEX IN GROUP I & II

	Clinical evidence of heart disease	No clinical evidence of heart disease
Male	4	13
Female	12	1

Pearson chi-square $X^2 = 14.0$ P = 0.001

When comparing Group I & Group II there is obvious sex difference, which is also statistically significant, more males in Group I, more females in Group II.

Out of 30 patients , 16 had clinical evidence of heart disease.

Cardiac Abnormality	Number	Percentage
Rheumatic heart disease	11	69%
Coronary artery disease	2	12.5%
Prosthetic valve	3	18.5%

This table shows rheumatic heart disease accounts for 11 cases (69%)of patients with heart disease. Still rheumatic heart disease forms the major cause of cardioembolic stroke in developing countries.

Three patients had prosthetic valve in mitral position . They were on oral anticoagulant therapy.

In all the 30 patients subjected to transthoracic echocardiography the findings are:

Rhematic heart disease	11
Mitral valve prolapse	2
Coronary heart disease	3
RHD with Left atrial clot	1
RHD with Left atrial spontaneous echocontrast	2
Prosthetic valve	3

Rheumatic heart disease

Based on associated valve involvement and rhythm disturbance the patients with rheumatic heart disease were further subdivided as shown below.

Isolated mitral valve involvement 5 → [4with AF], [1without AF]

Mitral and Aortic valve involvement 5 → [3with AF], [2without AF]

Mitral valve involvement with Left atrial clot - 1

Mitral, Aortic valve and Tricuspid valve - 1

In the study isolated mitral stenosis constitutes the predominant lesion which accounts for 45% (5 cases); in association with aortic valve involvement forms another 45% (5 cases); associated with tricuspid valve involvement in 10% (1 case).

Mitral stenosis is commonly associated with atrial fibrillation, which increases risk of stroke by 17fold compared with matched controls.

Only one case of rheumatic mitral stenosis had left atrial clot. In the remaining it is likely that fresh clots could have been discharged to systemic circulation.

Mitral valve prolapse

In 30 cases 2 (6%) cases were identified to have mitral valve prolapse by transthoracic echocardiography. Both the patients had no clinical evidence of heart disease i.e. they belonged to Group II.

Coronary artery disease

Two male patients below 40 yrs had ECG showing old inferior wall myocardial infarction. In both cases Transthoracic echocardiography showed hypokinetic inferior and posterior segments. One had associated left ventricular aneurysm with an adherent thrombus.

Based on Transoesohageal echocardiography

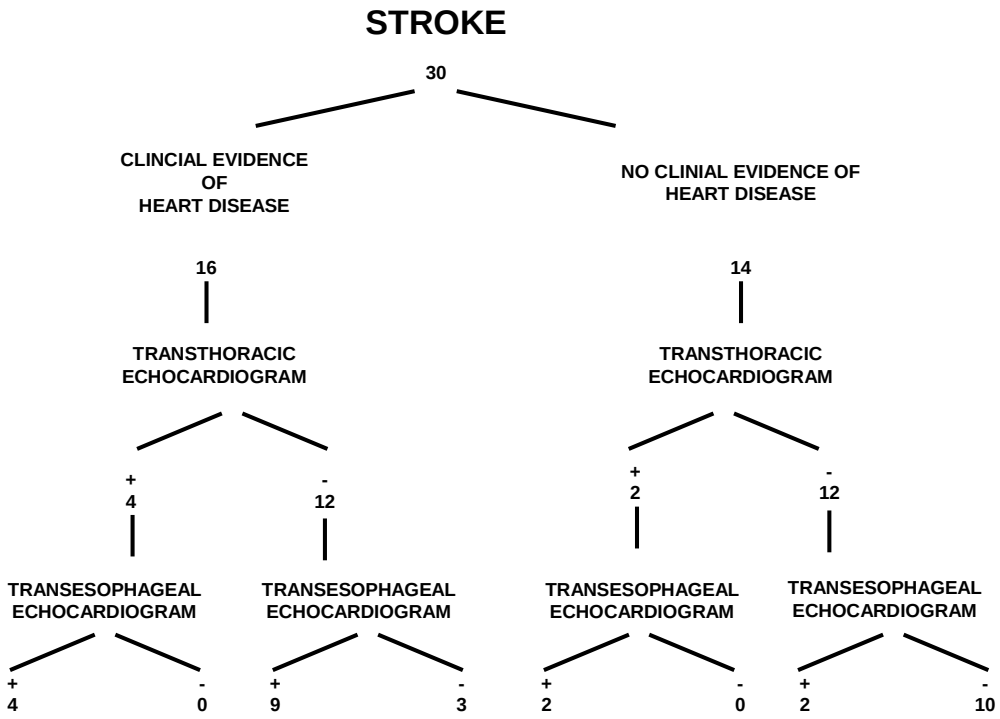
30 patients subjected to transoesophageal echocardiography findings are

Cardiac abnormality	Detected by TEE
Left atrial spontaneous echo contrast	8
Left atrial appendage clot	3
Left ventricular clot	1
Myoxmatous mitral valve with mitral regurgitation	2
Patent foramen ovale	1
Atrial septal aneurysm	1
Complex aortic atheroma	-
Prosthetic valve dysfunction	1

When comparing with transthoracic echocardiography transesophageal echocardiography additionally detected 6 cases of left atrial spontaneous echo contrast, 2 cases of left atrial appendage clot, one case of patent foramen ovale with atrial septal aneurysm, one case of prosthetic valve dysfunction.

Transesophageal echocardiography versus transthoracic echocardiography

The heart disease of patients in Group I was confirmed by transthoracic echocardiography. Transthoracic echocardiography also detected 1 left atrial clot in a patient with rheumatic mitral stenosis, left atrial auto contrast in 2 cases; left ventricular thrombus in 1 case. Totally it detected cardiac source of embolism in 4 cases (25%).



Transesophageal echocardiography revealed a potential cardiac source of embolism in 13patients (81%) i.e. it confirmed transthoracic echocardiography findings in 4 cases. And TEE detected an additional of 9 cases (56%) ,with left atrial spontaneous echo contrast in 6 cases (37 %), 2 more cases of rheumatic mitral stenosis showed left atrial thrombus, left atrial appendage being better visualised in transesophageal echocardiography. In one case of prosthetic valve, mild dysfunction was visualised in transesophageal echocardiography, out of 16 cases; 7 cases of RHD had atrial fibrillation and one patient with prosthetic valve had atrial fibrillation.

Group II cases, no clinical evidence of heart disease transthoracic echocardiography detected 2 cases (14%) of mitral valve prolapse which is also confirmed by transesophageal echocardiography and additionally detected 2

(14%) abnormalities ; one had patent foramen ovale detected by right to left shunting diagnosed by contrast studies and color flow imaging. This case also had associated atrial sepal aneurysm, which is an common accompaniment patent foramen ovale.

Statistical analysis

Detection rates of TTE statistically compared in those patients with clinical evidence of heart disease and in those with no clinical evidence of heart disease.

TTE

	Cardiac abnormalities present	**Cardiac abnormalities Not present**
Clinical evidence of heart disease	4 (25%)	12
No clinical evidence of heart disease	2 (14%)	12

Pearson chi-square X^2 Yates = 0.08 P = 0.78

It shows that difference is statistically not significant. However in patients with clinical evidence of heart disease, it is able to detect one out of four cases; whereas in no clinical evidence of heart disease one out of six cases.

TEE

Similarly detection rates of TEE statistically compared, with clinical evidence of heart disease in those with no clinical evidence of heart disease

	Cardiac abnormalities present	**Cardiac abnormalities Not present**
Clinical evidence of heart disease	13 (81%)	3
No clinical evidence of heart disease	4 (29%)	10

Pearson chi-square X^2 Yates = 6.43 P = 0.01 Odds ratio (O.R)=11(2-92)

It shows that difference is statistically significant, observed according to Yates corrected chi-square test. And also it shows that those with clinical evidence of heart disease TEE is able to detect 11 times more than those with no clinical evidence of heart disease.

TTE Vs TEE in patients with clinical evidence of heart disease.

	Cardiac abnormalities present	**Cardiac abnormalities Not present**
TTE	4	12
TEE	13	3

Pearson chi-square X^2 Yates = 8.03 P = 0.01 Odds ratio (O.R)=13 (2-108)

Comparing TEE against TTE in patients with clinical evidence of heart disease, cardiac abnormalities detected by TEE was 81% (13/16) patients against TTE which detected 25% (4/16) patients. It is statistically significant.

TTE Vs TEE in patients with no clinical evidence of heart disease.

	Cardiac abnormalities present	**Cardiac abnormalities Not present**
TTE	2	12
TEE	4	10

Pearson chi-square X^2 Yates = 0.21 P = 0.65 Not significant

Comparing TEE against TTE in patients with no clinical evidence of heart disease, cardiac abnormalities detected by TEE was 25% (4/14) patients against TTE which detected 14% (2/14) patients. This is not statistically significant.

Thoracic aorta was carefully examined in multiplane transesophageal echocardiography in all patient, no protruding thrombus was identified, none of them had atrial fibrillation in this group.

Out of 30 cases 17 (56%) patients ultimately had cardiac source of embolism identified by mean transesophageal echocardiography 22 (73%) in sinus rhythm and 8 had (27%) atrial fibrillation. Out of eight, seven patients with atrial fibrillation with potential cardial source of embolism detected. The most common cardiac source identified was spontaneous echocontrast. This abnormality is better detected by transesophageal echocardiography.

Cardiac abnormality	**Detected by TTE**	**Detected by TEE**	**Total**
Left atrial spontaneous echo contrast	2	8	8
Left atrial appendage clot	1	3	3
Left ventricular clot	1	1	1
Myoxmatous mitral valve with mitral regurgitation	2	2	2
Patent foramen ovale	-	1	1
Atrial septal aneurysm	-	1	1
Complex aortic atheroma	-	-	-
Prosthetic valve dysfunction	-	1	1

Left atrial spontaneous echo contrast was detected by TTE in 14% patients (2/16)with clinical evidence of heart disease, whereas TEE detected the same in 50%(8/16)patients. So detection rate of TEE against TTE is statistically significant (X^2 = 5.24, P=0.02, OR = 7 (1-63) according to Pearson chi-square test and hence TEE is 7 times more useful compared to TTE.

DISCUSSION

This study demonstrates the increased yield of transesophageal echocardiography in the detection of a potential cardiac source of embolism in stroke patients compared with transthoracic echocardiography.

In our study transthoracic echocardiography revealed a potential cardiac source of embolism in 25% (4 patients) all of whom had clinical evidence of heart diseases and 14% (2 patients) who had no clinical evidence of heart disease.

In addition to the yield by transthoracic echocardiography, transesophageal echocardiography revealed a potential source of embolism in 56% (9 cases). In patients with clinical evidence of heart disease, cardiac abnormalities additionally detected by transesophageal echocardiography are 2 cases of LA appendage clot, 6 cases of left atrial spontaneous echo contrast and one case of prosthetic valve dysfunction. In patients with no clinical evidence of heart disease, transesophageal echocardiography detected one case of patent foramen ovale with atrial septal aneurysm.

This is in keeping with previous studies[30,42,22]. **Pop et al.**[30] found 5 (9%) of 53 patients with recent TIA or stroke and without clinical cardiac abnormalities had abnormal TEE . Six (32%) of 19 patients with clinically suspected cardiac source of embolism had both a positive TEE and a positive TTE. The lower yield of TEE in that study may be due to lack of color flow imaging and contrast studies to detect intracardiac shunts. The proportion of patients with AF is not mentioned and was likely to be lower than in our study

Zenker et al.[40] found that 9 (45%) of 20 patients with a cerebral ischemic events under the age of 45 yrs and a normal TTE, TEE showed pathological findings which consisted mainly mitral valve prolapse, atrial septal defect and atrial aneurysms.

In the European multicenter study reported by **Daniel et al**[2]**.** 479 patients with unexplained arterial embolism were studied with TTE and TEE. The majority of these patients had a cerebral ischemic event. Potential source of arterial source of embolism were detected by TTE in 176 (37%) of 479 patients and by TEE in 310 (65%) of 479 patients. TEE identified mitral valve prolapse, patent foramen ovale, left atrial and atrial appendage thrombi, spontaneous echocontrast, atrial septal aneurysm and valvular vegetation significantly more frequently than transthoracic echocardiography. The yield of TEE was higher in that study because of the inclusion of patient with peripheral arterial embolism.

Black et al[24]**.** 100 patients with cerebral ischemic event or peripheral arterial embolism (63), before percutaneous Balloon dilatation of mitral valve (23), or before electrical cardioversion (14) were studied with TTE and TEE. TEE showed potential embolic sources in 36/53 (68%) patients with AF compared with 9/47 (19%) with sinus rhythm. TEE identified left atrial spontaneous echocontrast, left ventricular spontaneous echocontrast, mitral valve prosthesis thrombus, mitral valve prolapse and pronounced aortic atheroma significantly and more frequently than TTE, which helped in avoidance of Balloon dilatation of mitral valve in some patients.

Cujec et al[10]**.** compared diagnostic yields of TEE and TTE in detection of cardiac source of embolism. 63 patients with TIA or stroke underwent both procedures, TTE revealed potential source of cardiac embolism in 14% (9) of the patients with clinical evidence of heart disease, TEE revealed a potential cardiac source of embolism in 41% (26) of the patients, 27% (7) of these patients has no clinical cardiovascular abnormalities. Abnormality detected only by TEE included atrial septal aneurysm, PFO, left atrial appendage thrombus and myxomatous mitral valve. Patient identified cardiac source of embolism more frequently in AF and had larger left atrium.

	TTE	**TEE**
Pope et al.	Clinical evidence heart disease 6/19 (32%)	6/19 (32%)
	Normal heart	5/53 (9%)
Daniel et al.	176/479 (37%)	310/474 (65%)
Black et al.		36/53 (68%) 9/47 (19%)
Cujec et al.	Normal	7/39 (18%)
	Clinical evidence heart disease 9/20 (38%)	19/24 (79%)
Present study	Normal 2/14 (14%)	4/14 (25%)
	Clinical evidence heart disease 4/16 (25%)	13/16 (81%)

Our present study correlates well with Cujec et al. both for no clinical evidence of heart disease (X^2=1.54, P=0.21; Not significant) and clinical evidence of heart disease (X^2=0.03, P=0.87; Not significant); because there is no statistical significant difference.

In our study TTE revealed only mitral valve prolapse in patients without clinical evidence of cardiac disease. This is in keeping with previous reports. In 25% of the patients with clinical evidence of heart disease, a potential source of cardiac embolism was detected by TTE. The incidence of transthoracic echo cardiographic abnormalities in stroke patients varies from 4 to 47%[8,20] depending on the criteria used. A definitive source of embolism such as valvular vegetation or thrombus or myxoma are detected in very few patients.

Patient with clinical evidence of cardiac disease frequently have potential cardiac source of embolism detected by TEE. Although its diagnostic yield in patients without obvious heart disease is low, TEE may have therapeutic impact and should be performed in young patients or those with multiple stroke or systemic embolism.

TEE is superior to TTE for the detection of left atrial thrombus, patent foramen ovale, atrial septal aneurysm, left atrial spontaneous echocontrast and myxomatous degeneration of mitral valve, associated with mitral valve prolapse. All of these cardiac abnormalities may be a source of embolism.

Atrial fibrillation is a well known contributor to stroke. In our study, out of 8 patients in seven with atrial fibrillation transesophageal echo cardiogram revealed cardiac source of embolism. **Belder et al.**[4] found that patients in atrial fibrillation with left atrial spontaneous echocontrast are four times more likely to have suffered thromboembolic event than patients in AF without spontaneous echocontrast . Three of the patients with left atrial spontaneous echocontrast had left atrial appendage thrombus. In patients with AF underlying structural heart disease is ruled out by TEE, then it is more likely to be "lone AF".

One of the major limitations of this study is the lack of age matched control group of volunteers without strokes. The cardiac abnormalities associated with systemic embolism can also be detected in persons who do not have strokes, although the prevalence of all these abnormalities is higher in patients with strokes. Only stroke cases were taken for the study. Patients with TIA & normal CT brain were excluded. Saline contrast studies were not performed during TTE. The patient with PFO was detected by color flow imaging and contrast study only by TEE. Transthoracic contrast echo is insensitive and non specific for the detection of right to left shunting through patent foramen ovale.

CONCLUSION

The prevalence and pattern of cardiac disorder in 30 ischemic stroke patient was studied and analysed.

1. Transesophageal echocardiography is of great value in patients with clinical evidence of heart disease. In patients with no clinical evidence of heart disease transesophageal echocardiography has proved to be useful to detect lesions in small percentage of patients without clinical evidence of heart disease which are not even detected by transthoracic echocardiography. Hence transesophageal echocardiography is superior to transthoracic echocardiography in the detection of cardiac source of embolism in patients with cerebral ischemic event.

   Since this present study involves only small no. of patients usefulness of TEE in patients with no clinical evidence heart disease needs more studies involving large number of patients.

2. In this study Rheumatic heart disease stands as the foremost source of embolism (36%). Among this mitral valve involvement was found in almost all cases. The highest yield in TEE was associated with atrial fibrillation than without atrial fibrillation. Mitral valve prolapse accounted for 6% of cases of ischemic stroke. Coronary artery disease is an uncommon source of embolism in patients less than 45 yrs. Prosthetic valve is also an important source of embolism .

3. Clots in cardiac chambers was detected only in 10% of cases.

4. Patent foramen ovale was detected in 3% of cases.

SUMMARY

To compare the diagnostic yield of transesophageal echocardiography and transthoracic echocardiography in the detection of potential cardiac source of embolism in young stroke patients, 30 patients with stroke underwent both procedures. Transthoracic echocardiography revealed a potential cardiac source of embolism in 25% (4 patients) all of whom had clinical evidence of heart diseases and 14% (2 patients) who had no clinical evidence of heart disease.

In addition to the yield by transthoracic echocardiography, transesophageal echocardiography revealed a potential source of embolism in 56% (9 cases). In patients with clinical evidence of heart disease, cardiac abnormalities additionally detected by transesophageal echocardiography are 2 cases of LA appendage clot, 6 cases of left atrial spontaneous echo contrast and one case of prosthetic valve dysfunction (which is statistically significant). In patients with no clinical evidence of heart disease, transesophageal echocardiography detected one case of patent foramen ovale with atrial septal aneurysm. Thus transesophageal echocardiography is more sensitive than transthoracic echocardiography in detection of potential cardiac source of embolism.

PROFORMA

CARDIAC SOURCE OF EMBOLISM IN YOUNG STROKE

Name	Age	Sex	I.P.No.
D.O.A.	D.O.D.	Unit	

Complaints **Duration**

Weakness of limb Rt/Lt
Aphasia
Headache
Vomiting
Seizures
Loss of consciousness
Previous stroke
Others

Past history

DM
Hypertension
Smoking
Prev heart disease
Oral anti coagulant
Hyper cholesterolemia
Apla/Prothrombotic states
Alcoholism
Collagen vascular disease
OC Pills
Others
TIA

General Examination

Anemia
Cyanosis

Clubbing
Lctrus
Lymphadenopathy
Pedal oedema
Pulse
Blood pressure
R.R.

CVS

RS

ABDOMEN

CNS

INVESTIGATIONS

Hb	TC	DC	ESR			
SUGAR	UREA	CREAT	Na	K	Cl	
VDRL	HIV					
Sr. LIPID PROFILE						
RF	CRP	ANA				
OTHERS						

ECG

Rate	axis	AF	
QRS	RVH	LVH	Crochetage
OTHERS			

Chest X-ray

ECHO

CD No Echo No.

TTE	**TEE**
LA spont echocontrast	LA spontaneous echo contrast
LA thrombus	LA Thrombus
LVH	Complex aortic atheroma >5mm
LV apical thrombus	Patent foramen ovale
Valvular heart disease	Atrial Septal aneurysm
MS	Valvular vegetations & intra cardiac masses
AS	Mitral valve prolapse
MR	
TR	
Mitral valve calcification	

PHT

LV wall motion abnormalities N/AB

Hypokinetic Akinetic

LV Aneurysm

LV wall scaring

LV systolic function

Mitral valve prolapse

CT BRAIN	**MRI BRAIN**

ABBREVIATIONS

AF	-	Atrial fibrillation
AR	-	Aortic regurgitation
ASA	-	Atrial septal aneurysm
AS	-	Aortic stenosis
AMI	-	Acute myocardial infarction
CMC	-	Closed mitral commissurotomy
CT	-	Computed tomography
CVA	-	Cerebrovascular accident
IHD	-	Ischemic heart disease
IWMI	-	Inferior wall myocardial infarction
LVH	-	Left ventricular hypertrophy
LAE	-	Left atrial enlargement
LA	-	Left atrium/al
LV	-	Left ventricle/ventricular
MS	-	Mitral stenosis
MVP	-	Mitral valve prolapse
MCA	-	Middle cerebral artery
MR	-	Mitral regurgitation
NSR	-	Normal sinus rhythm
PFO	-	Patent foramen ovale
RBBB	-	Right bundle branch block
RVH	-	Right ventricular hypertrophy
RVR	-	Rapid ventricular response
RHD	-	Rheumatic heart disease
TTE	-	Transthoracic echo cardiography
TEE	-	Transesophageal echo cardiography
WNL	-	Within normal limits

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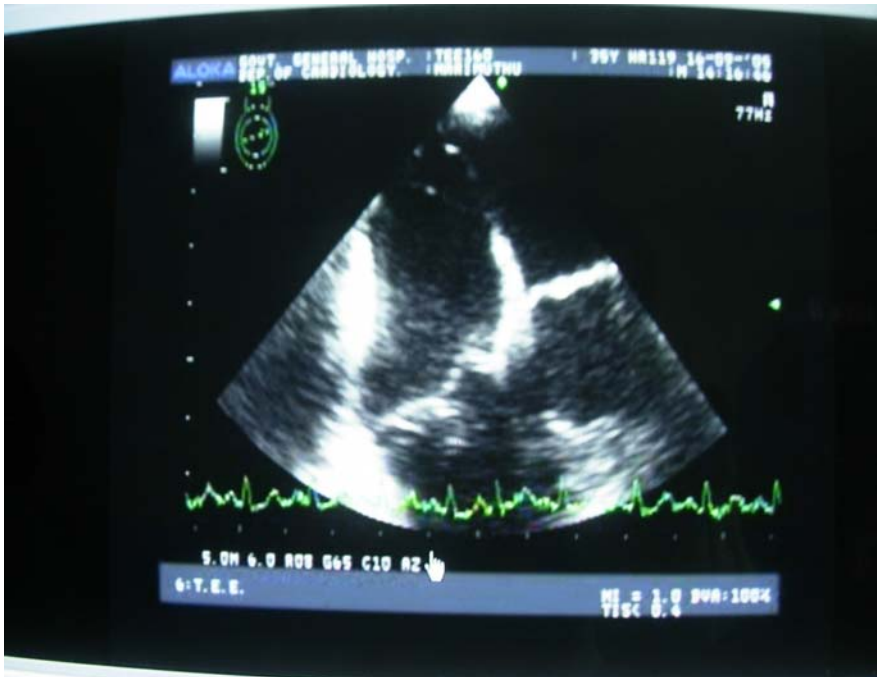
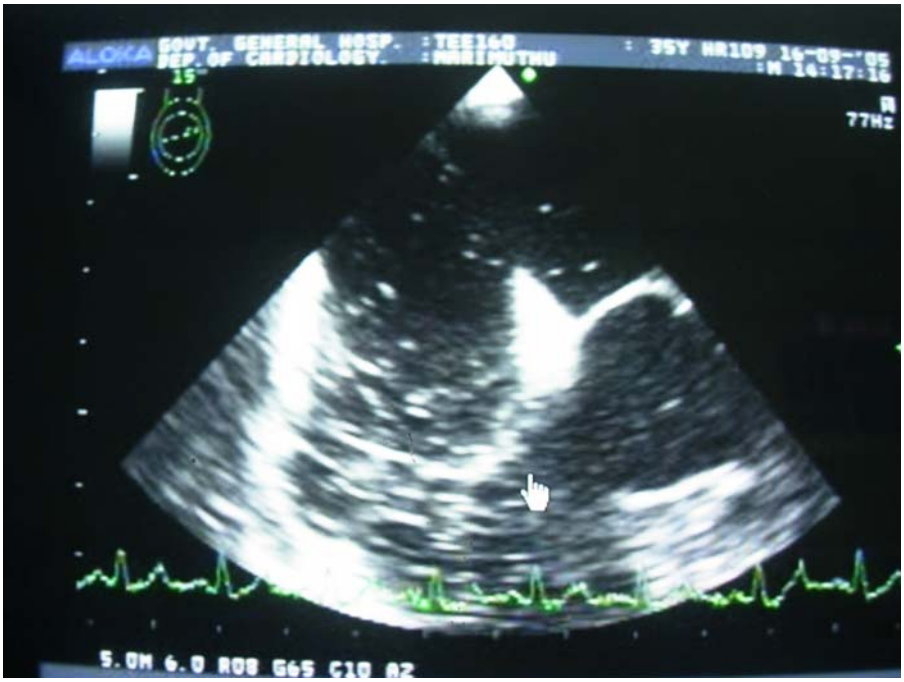
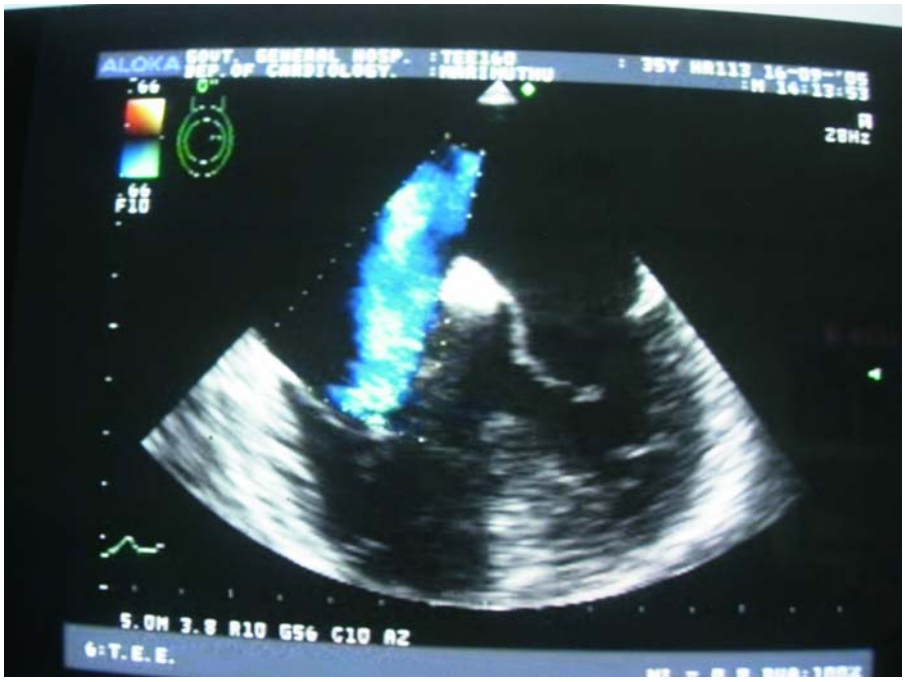


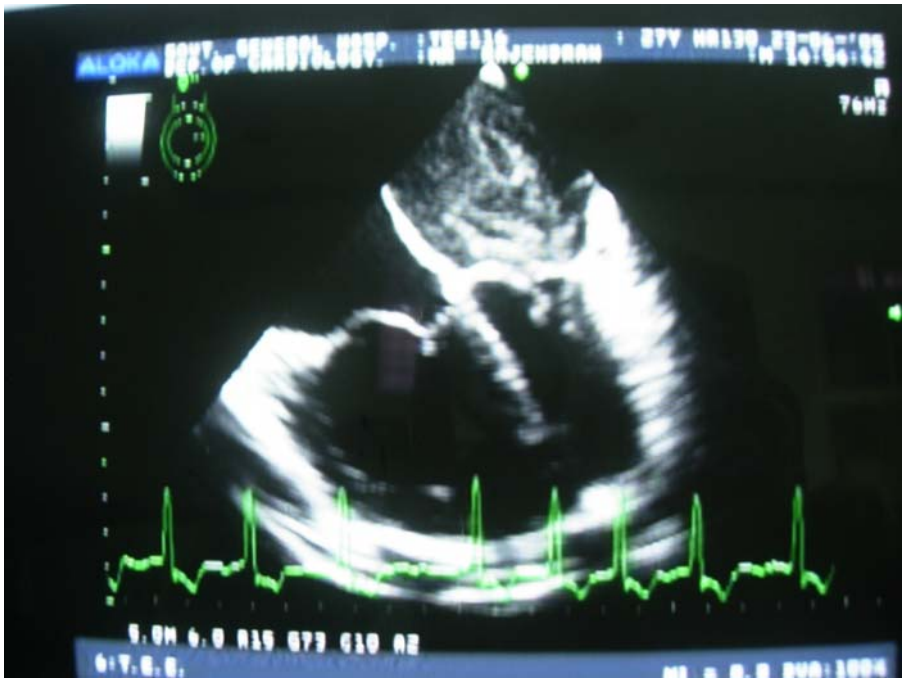
TEE showing PFO with atrial septal aneurysm



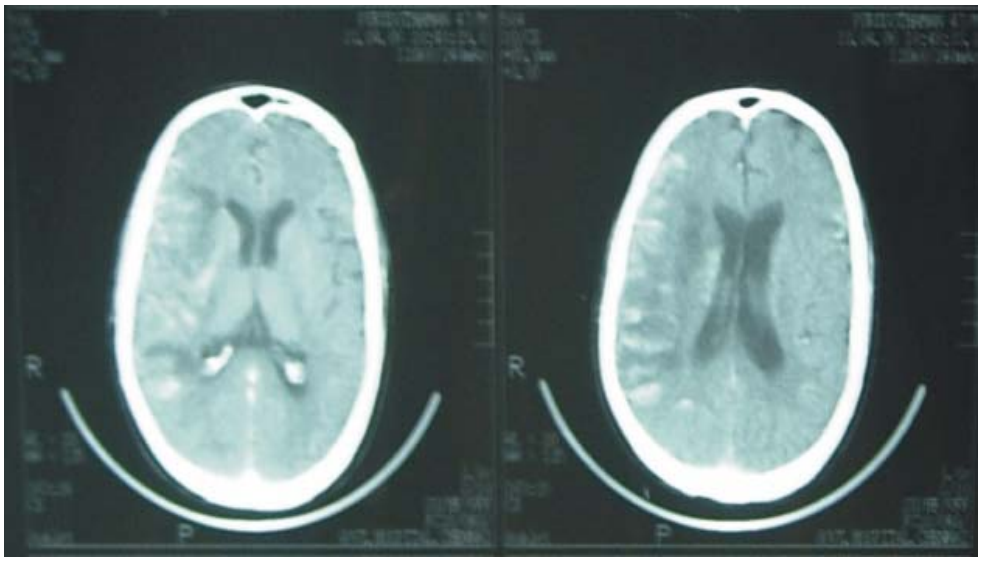
TEE showing opacification of right atrium and saline contrast cross over to left atrium



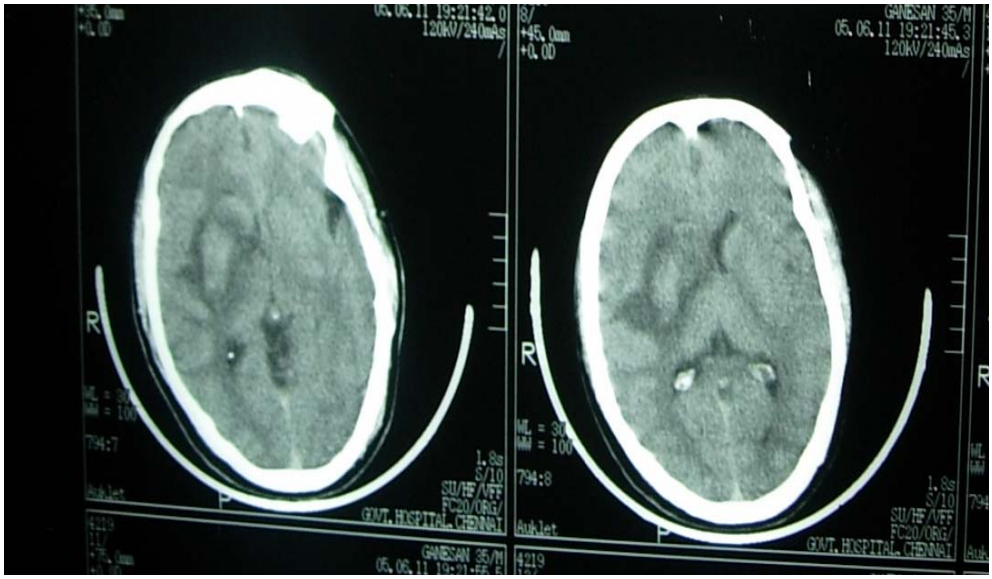
TEE bicaval view color Doppler showing flow across the inter atrial septum



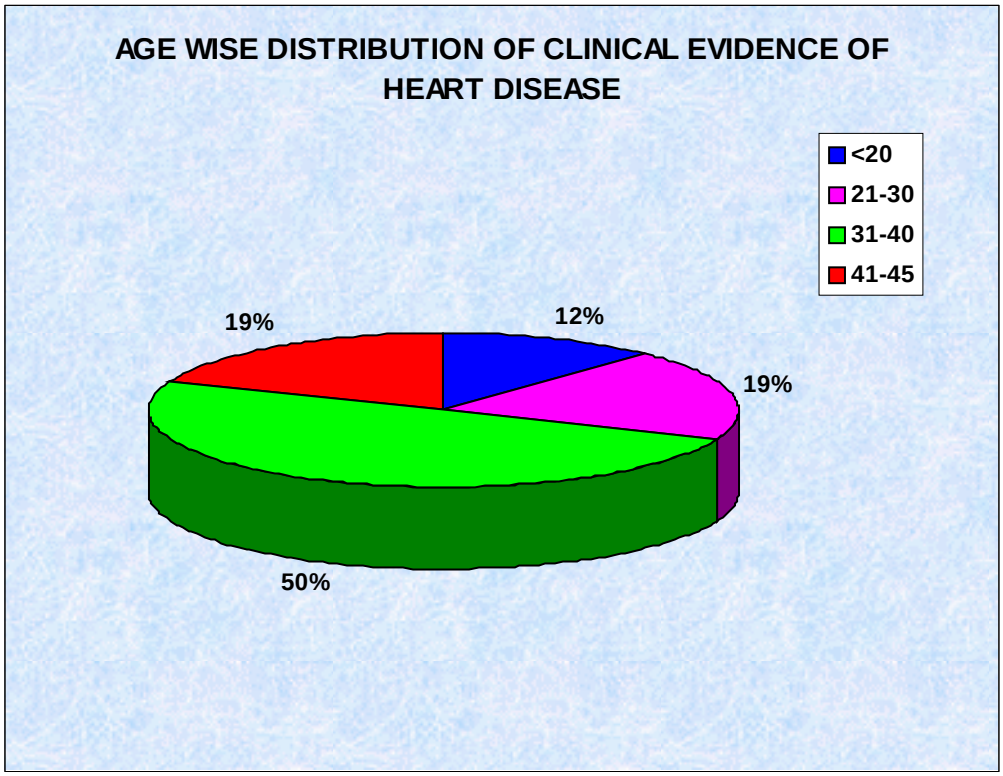
TEE apical four chamber view showing LA spontaneous echo contrast with LA appendage clot

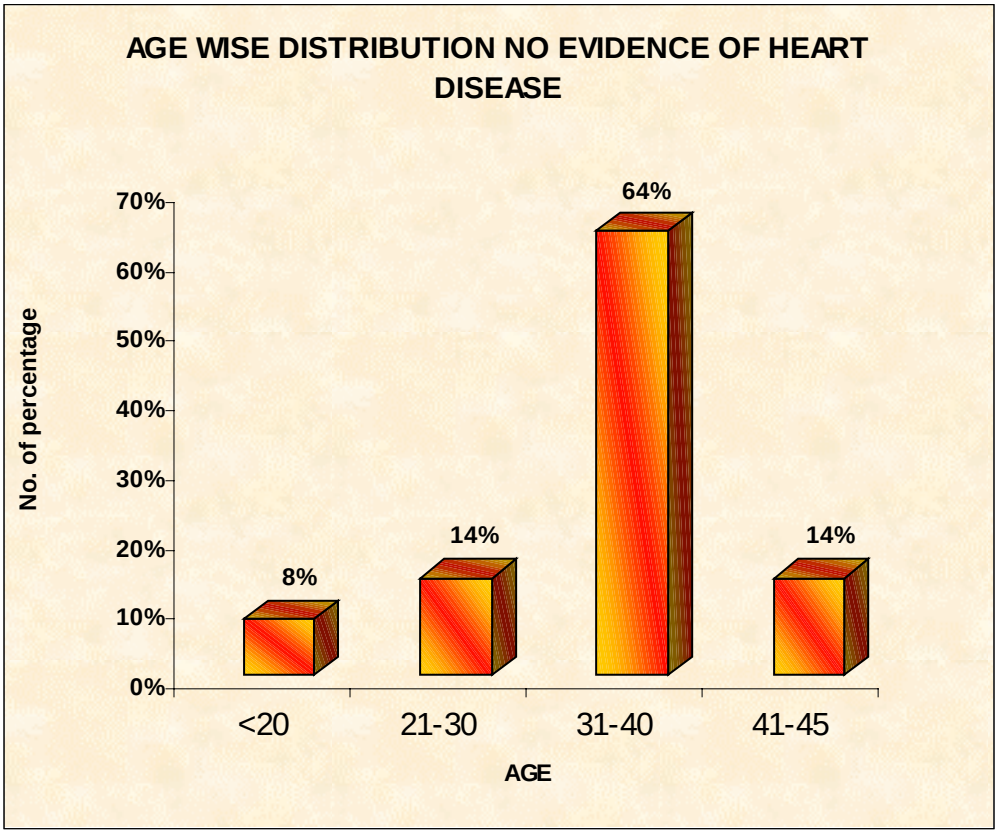


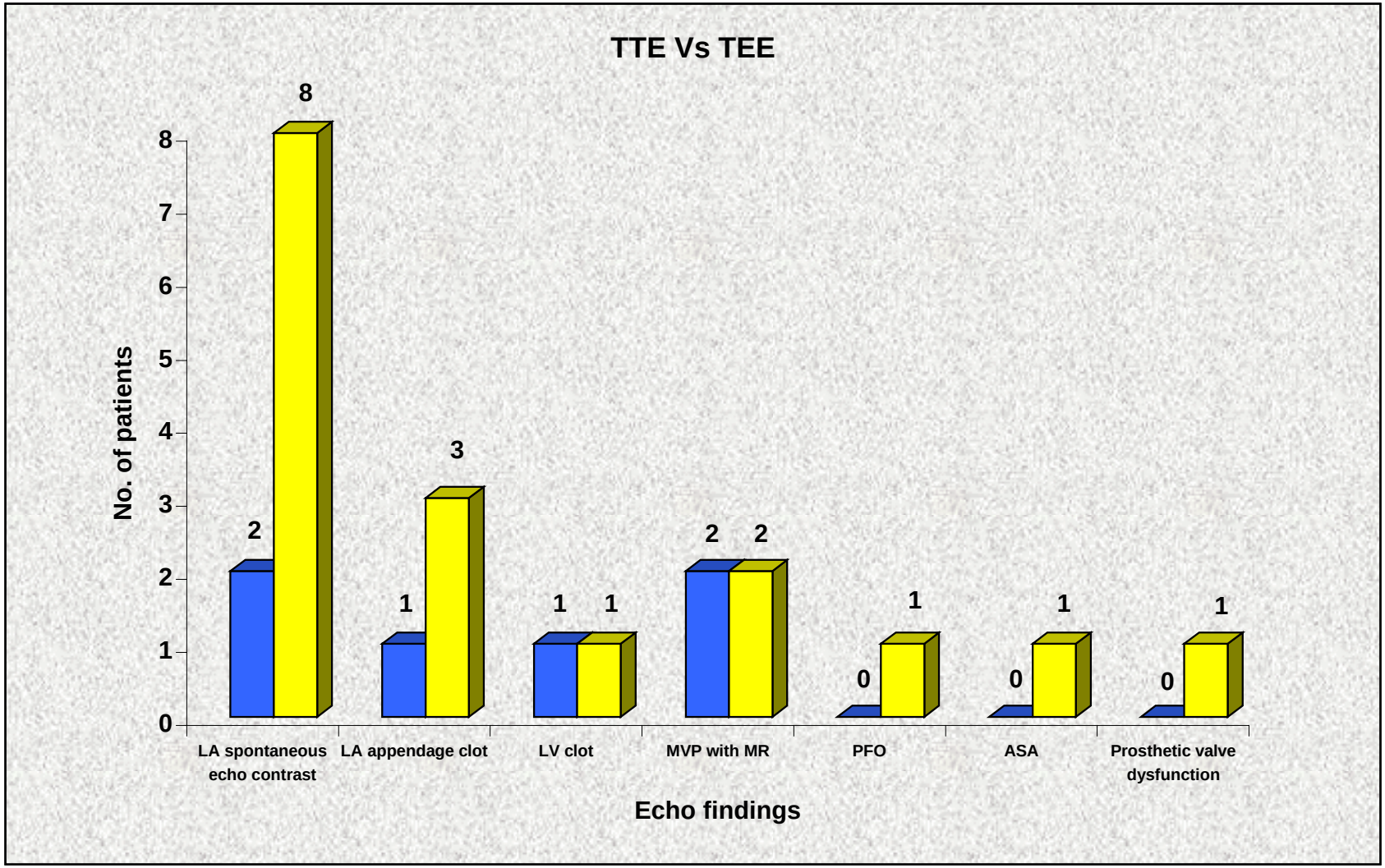
CT brain (plain) axial section showing right MCA territory infarct with hemorrhagic transformation



CT brain (plain) axial section showing right MCA territory infarct with hemorrhagic transformation


AGE WISE DISTRIBUTION OF CLINICAL EVIDENCE OF HEART DISEASE
<20
21-30
31-40
41-45
19%
12%
19%
50%


AGE WISE DISTRIBUTION NO EVIDENCE OF HEART DISEASE
No. of percentage
70%
60%
50%
40%
30%
20%
10%
0%
8%
14%
64%
14%
<20
21-30
31-40
41-45
AGE


TTE Vs TEE
No. of patients
0
1
2
3
4
5
6
7
8
2
8
1
3
1
1
2
2
0
1
0
1
0
1
LA spontaneous echo contrast
LA appendage clot
LV clot
MVP with MR
PFO
ASA
Prosthetic valve dysfunction
Echo findings

PERCUTANEOUS DILATATIONAL TRACHEOSTOMY GROUP

Sl. No.	Name, Age, Sex, IP No. Ward	No. of days intubated	Diagnosis	Duration of procedure in min.	Intraoperative complications							Post-operative Complication								Follow up	
					Lowest SaO_2	Major Bleed	Minor Bleed	False Passage	Resistant to Insertion	Transient hypoten.	Transient hypoxia	Major bleed	Minor bleed	Stomal infec.	Pneumo. thorax	Death	Sub. emphy.	TE fistula	TA fistula	Deccanulated	Tra. Stenosis
1.	Narayanan 36/M 633301 N1 wd.	7	Extradural haemorrhage	12	94	-	-	Yes	-	-	-	-	-	-	-	-	-	-	-	Yes	-
2.	Sekar 19/M 635070 N1 wd.	12	Lt temparo parietal contusion	9	90	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3.	Selvakumar 22/M 635428 N1 wd.	6	Subdural haemorrhage	8	94	-	-	-	-	-	Yes	-	-	-	-	-	-	-	-	-	-
4.	Neela 60/F 637664 IMCU	9	Brainstem infarct	10	96	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
5.	Parasuraman 56/M 637519 IMCU	12	Type II DM with Necrotising pneumonia	12	95	-	-	Yes	-	-	-	-	-	-	-	-	-	-	-	-	-
6.	Raju 18/M 635419 IMCU	11	Guillain barre syndrome	13	99	-	Yes	Yes	-	-	-	-	-	-	-	-	-	-	-	Yes	-
7.	Manjunathan 45/M 638471, N1 Wd	6	Extradural haematoma craniotomy done	8	90	-	-	-	-	-	Yes	-	-	-	-	-	-	-	-	-	-
8.	Sakthivelu 58/M 639162 IMCU	10	Type 2 DM with pulm HT	10	92	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
9.	Karvan 34/M 643857 IMCU	7	Brainstem stroke	11	96	-	-	-	Yes	-	-	-	-	-	-	-	-	-	-	-	-
10.	Peter 46/M 668254 IMCU	8	HT with DM with Hypoxic encephalopathy	7	93	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
11.	Mr.Vasudevan 45/M 674125 N1 Wd	7	Left parietal SDH	10	94	-	-	-	-	Yes	-	-	-	-	-	-	-	-	-	-	-
12.	Mr. Krishnakumar	6	Rt. temparo parietal	10	98	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Sl. No.	Name, Age, Sex, IP No. Ward	No. of days intubated	Diagnosis	Duration of procedure in min.	Intraoperative complications							Post-operative Complication								Follow up	
					Lowest SaO_2	Major Bleed	Minor Bleed	False Passage	Resistant to Insertion	Transient hypoten.	Transient hypoxia	Major bleed	Minor bleed	Stomal infec.	Pneumo. thorax	Death	Sub. emphy.	TE fistula	TA fistula	Deccanulated	Tra. Stenosis
	30/M 678599 N1 Wd		contusion																		
13.	Rajan 42/M 678486 N1 Wd	10	Brainstem hemorrage	8	97	-	-	-	-	Yes	-	-	-	-	-	-	-	-	-	-	-
14.	Thangaraj 24/M 680101 IMCU	8	OPC poisoning	12	96	-	Yes	-	-	-	-	-	-	-	-	-	Yes	-	-	-	-
15.	Arumaidoss 30/M 680181 IMCU	6	Guillain Barre syndrome	6	91	-	-	-	-	-	-	-	-	-	-	-	Yes	-	-	-	-
16.	Venkatesan 70/M 680108 N1 Wd	5	Intracerebral hemorrahage	10	92	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
17.	Ganesh 24/M 680204 N1 Wd	7	SDH & EDH Operated	7	94	-	-	-	-	Yes	-	-	-	-	-	-	-	-	-	-	-
18.	Hemavaty 21/F 673344 IMCU	7	OPC Poisioning	10	90	-	-	-	-	-	-	-	-	-	-	-	-	-	-	Yes	-
19.	Jayalakshmi 40/F 680291 N1 Wd	7	CP Angle tumour operated	8	90	-	-	-	-	-	-	-	-	Yes	-	-	-	-	-	-	-
20.	Selvam 30/M 680201 IMCU	8	OPC poisoning	15	98	-	-	Yes	-	-	-	-	-	-	-	-	-	-	-	-	-
21.	Chandrasekar 45/M 672282 IMCU	7	RTA with multiple injuries	8	94	-	-	-	-	-	-	-	Yes	-	-	-	-	-	-	-	-
22.	Balakrishnan 35/M 680095 N1 Wd	7	Intracerebral hemorrage	10	92	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
23.	Natarajan 48/M 679575 N1 Wd	11	RTA with multiple contusion brain	7	90	-	-	-	-	-	Yes	-	-	-	-	-	-	-	-	-	-
24.	Sampathkumar 34/M 680120 N1 Wd	9	RTA Rt. frontoparietal contusion	9	90	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
25.	Murugan 37/M 680185	6	RTA with Temporal	10	97	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Sl. No.	Name, Age, Sex, IP No. Ward	No. of days intubated	Diagnosis	Duration of procedure in min.	Intraoperative complications							Post-operative Complication								Follow up	
					Lowest SaO_2	Major Bleed	Minor Bleed	False Passage	Resistant to Insertion	Transient hypoten.	Transient hypoxia	Major bleed	Minor bleed	Stomal infec.	Pneumo. thorax	Death	Sub. emphy.	TE fistula	TA fistula	Deccanulated	Tra. Stenosis
	N1 Wd		Depressed fracture																		

STANDARD OPERATIVE TRACHEOSTOMY GROUP

Sl. No.	Name, Age, Sex, IP No. Ward	No. of days intubated	Diagnosis	Duration of procedure in min.	Intraoperative compucations							Post-operative Compucation								Follow up	
					Lowest SaO_2	Major Bleed	Minor Bleed	False Passage	Resistance to Insertion	Trasient hypoten.	Trasient hypoxia	Major bleed	Minor bleed	Stomal infec.	Pneumo. thorax	Death	Sub. emphy.	TE fistula	TA fistula	Deccanulated	Tra. Stenosis
1.	Sakunthala 23/F 632663 IMCU	7	Encephalitis	20	98	-	-	-	-	-	Yes	-	-	-	-	-	-	-	-	-	-
2.	Anbudurai 22/M 632603 N1 Wd	5	RTA with Frontoparietal contusion	25	94	-	Yes	-	-	-	-	-	-	Yes	-	-	-	-	-	-	-
3.	Raju 18/M 635419 IMCU	10	Transverse myelitis	22	96	-	-	-	-	-	-	-	Yes	-	-	-	-	-	-	Yes	-
4.	Vimala 42/F 636205 IMCU	8	COPD with respiratory failure	25	90	Yes	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
5.	Kamalam 62/F 637427 N1 Wd	6	RTA with extradural hemorrhage	25	97	-	Yes	-	-	-	-	-	-	-	-	-	-	-	-	Yes	Yes
6.	Mohammed Ali 21/M 636508 N1 Wd	9	RTA with intracerebral hemorrhage	20	92	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
7.	Venkatesan 22/M 638997 IMCU	7	RHD with pulmonary edema	30	97	Yes	-	-	-	-	-	-	Yes	-	-	-	-	-	-	Yes	-
8.	Manikandan 25/M 639022 N1 Wd	7	RTA with occipital contusion	18	95	-	-	-	-	-	Yes	-	-	-	-	-	-	-	-	Yes	-
9.	Kesavaraj 39/M 638839 N1 Wd	6	RTA with frontal contusion with intracerebral hemorrhage	24	98	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
10.	Govindhammal 28/F 639812 IMCU	7	RTA with Brainstem dysfunction	26	90	-	Yes	-	-	-	-	-	Yes	Yes	-	-	-	-	-	-	-
11.	Thulasi 50/F 639377 N1 Wd	12	RTA Rt Fronto parietal contusion	30	88	Yes	-	-	-	-	-	-	Yes	-	-	-	-	-	-	-	-

Sl. No.	Name, Age, Sex, IP No. Ward	No. of days intubated	Diagnosis	Duration of procedure in min.	Intraoperative compucations							Post-operative Compucation								Follow up	
					Lowest SaO_2	Major Bleed	Minor Bleed	False Passage	Resistance to Insertion	Trasient hypoten.	Trasient hypoxia	Major bleed	Minor bleed	Stomal infec.	Pneumo. thorax	Death	Sub. emphy.	TE fistula	TA fistula	Deccanulated	Tra. Stenosis
12.	Iqbal 25/M 640216 IMCU	6	OPC poisoning	28	99	-	-	-	-	-	-	-	Yes	-	-	-	-	-	-	Yes	-
13.	Sampath 51/M 662702 IMCU	8	CVA with Rt intracerebral hemorrhage	25	94	-	Yes	-	-	-	-		-	Yes	-	-	-	-	-	-	-
14.	Kiran 22/M 664939 N1 Wd	8	RTA with Lt frontal compound depressed fracture	20	97	-	-	-	-	Yes	-	-	-	-	-	-	Yes	-	-	-	-
15.	Nayagam 50/F 665978 N1 Wd	5	RTA Lt Temparoparietal SDH	26	90	-	Yes	-	-	-	-	-	-	-	-	-	-	-	-	-	-
16.	Balasubramanian 54/M 6665104,IMCU	7	RTA with multiple injuries	22	92	-	-	-	-	-	-	-	-	Yes	-	-	-	-	-	Yes	-
17.	Balakrishnan 52/M 680811 N1 Wd	5	RTA with extradural hemorrhage	18	97	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
18.	Muthammal 50/F 681212 N1 Wd	7	RTA intracerebral hemorrhage	26	99	-	Yes	-	-	-	-	Yes	-	-	-	-	-	-	-	-	-
19.	Ramzan Bevi 50/F 682227 IMCU	10	Myaesthenia gravis	25	90	-	-	-	-	Yes	-	-	-	Yes	-	-	-	-	-	Yes	-
20.	Gurunathan 50/M 681354 N1 Wd	7	RTA with subdural hemorrage	28	92	-	Yes	-	-	-	-	-	-	-	-	-	Yes	-	-	-	-
21.	Paneerselvam 55/M 683795 N1 Wd	7	RTA with Lt temparo parietal contusion	25	92	-	-	-	-	-	-	-	-	-	-	-	-	-	-	Yes	-
22.	Ashok Kumar 22/M 684474 N1 Wd	8	RTA Rt fronto parietal contusion	25	94	-	-	-	-	-	-	-	-	Yes	-	-	-	-	-	-	-
23.	Murugesan 28/M 684810 N1 Wd	6	RTA Rt fronto parietal contusion	20	93	-	Yes	-	-	-	-	-	-	Yes	-	-	-	-	-	-	-
24.	Baby Albert 28/M	7	TB meningitis	24	97	-	-	-	-	-	-	-	-	-	-	-	-	-	-	Yes	-

Sl. No.	Name, Age, Sex, IP No. Ward	No. of days intubated	Diagnosis	Duration of procedure in min.	Intraoperative compucations							Post-operative Compucation								Follow up	
					Lowest SaO_2	Major Bleed	Minor Bleed	False Passage	Resistance to Insertion	Trasient hypoten.	Trasient hypoxia	Major bleed	Minor bleed	Stomal infec.	Pneumo. thorax	Death	Sub. emphy.	TE fistula	TA fistula	Deccanulated	Tra. Stenosis
	638619 IMCU																				
25.	Nandhurayan 42/M 640716 N1 Wd	6	RTA with SDH	30	92	Yes	-	-	-	-	-	-	Yes	-	-	-	-	-	-	-	-