A STUDY OF CARDIAC DYSRHYTHMIAS

IN OLEANDER SEED POISONING .

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CERTIFICATE

This is to certify that DR. S.VENMATHI , postgraduate student (July 2003 to September 2006) in the Department of Internal Medicine , Kilpauk Medical College ,Chennai – 600 010 , has done this dissertation on " A STUDY OF CARDIAC DYSRRYTHMIAS IN OLEANDER SEED POISONING " under my guidance and supervision in partial fulfillment of the regulations laid down by The Tamilnadu Dr.M.G.R. Medical University, Chennai, for M.D. (General Medicine), Degree Examination to be held in September 2006.

Prof . D.S .SOMASEKAR , M.D.
Prof .of Medicine
Kilpauk medical college &
Govt. Royapettah Hospital,
Chennai.

Prof .K.S.Saikumar ,M.D.
Professor & HOD
Department of Internal Medicine
Kilpauk Medical College.
Chennai.

DR.THIYAGAVALLI KIRUBAKARAN M.D.
The Dean
Kilpauk Medical College
Chennai .

Date :15.04.2006.
Station :Chennai.

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ABBREVATIONS

SA	Sino atrial
AV	Atrio ventricular
RMP	Resting membrane potential.
ERP	Effective refractory period
NA-K ATPase	Sodium potassium ATPase
CTZ	Chemoreceptor trigger zone
CNS	Central nervous system
CVS	Cardiovascular system
RBS	Random blood sugar
RBC	Red blood cell
BUN	Blood urea nitrogen
LDH	Lactate dehydrogenase
ECG	Electrocardiogram
EPS	Electrophysiological study
AT	Atrial tachycardia
VT	Ventricular tachycardia
VPC	Ventricular premature complex
APC	Atrial premature complex
VF	Ventricular fibrillation
AF	Atrial fibrillation

CONTENTS

INTRODUCTION

In developing countries, the hospital admissions due to suicidal attempts are relatively high. Even in western countries mortality due to suicidal attempt is on the rise. The factors influencing suicidal attempts mainly depends on cultural &ethnic background. It is essential to prevent or decrease the number of deaths due to suicidal attempts.

In India the incidence of suicidal attempts is more in females. This may be attributed to gender discrimination , lower educational standards , Lack of financial & social independence . Various methods of suicidal attempts used are hanging ,drowning, ingestion of chemicals, corrosives or indigenous plant substances . Drug abuse or over dosage also contributes.

Among the indigenous plants Oleander is more commonly used. There are two types of oleander

i) Yellow Oleander -Cerebra thevetia.

ii) White Oleandar - Nerium odorum**.**

In our population Yellow oleander poisoning is more commonly seen. Yellow oleander is more potent & toxic than White oleander. All parts of the plant namely The Root, Stem, Twig, Leaf, Flower, Fruit, Kernel & Seed are poisonous. These plant parts have a high content of alkaloid whose action is similar to digitalis. Cardio toxicity is the major cause of death in oleander poisoning.

The proportion of hospital admissions due to oleander seed poisoning is high. The incidence is high in adolescence & early adult life. This group of population is productive, economically. suicidal attempts can be effectively prevented by comprehensive health care & counseling. Poisoning due to indigenous plants is more common in India which should be prevented or treated appropriately.

AIM OF STUDY

1. To determine the incidence of oleander seed poisoning among hospital admissions in Govt. Royapettah hospital from April 2004-August2004.

2. To elucidate the factors influencing the incidence of oleander seed poisoning,

a) Age.

b) Sex.

c) Education.

d) Socio economic status.

3.To analyze various factors influencing mortality in oleander seed poisoning

a)Variety of oleander.

b)Part of the plant consumed.

c)Amount consumed.

d)Mode of consumption.

e)Time window.

f)Cardiac Dysrrythmias.

HISTORICAL REVIEW:

Oleander plant was known since ancient times, finds mention in CHARAKA SAMHITA under 30 – 40 different names in Sanskrit. The name thevetia was given in honour to Mr. Andre Thevit who traveled extensively in South America during 16th century and who has written extensively about this plant.

All parts including smoke from burning cuttings and water in which the flower is placed are poisonous. The kernels of the seed are 8 times more poisonous than the leaves followed by latex of the plant. It was used as a suicidal or cattle poison. SHATAKUNDU meaning SHATA as hundred and KUNDU as hammer brings out the dangerous effects clearly. Other names include KARAVIRA, VIRAKA {fierce}, LAKUDA {weapon made of stick}, RAKTHA PUSHPA {blood flower} depicting its toxic nature.

From the above names we clearly understand that different parts of the plants were used rather abused in ancient times because of its lethal properties.

Yellow oleander finds its place among five deadly plant poisons included in UPAVISHA PANCHAKA, other 4 being SMUKI,

ARAKA, LANGALI, and KUCHELEKA. Arrows were poisoned with oleander extract by South American and east African tribes.

Reports about numerous deaths from oleander branches being used as a food skewer, from oleander smoke, and from flower were of historical interest as well as present interest.

REVIEW OF LITERATURE

Cerbera Thevetia

All parts of the plants are poisonous.

A: Flowers: They are large, bell shaped, yellow 57 cms long. Five lobes of the flower are spirally twisted and spreading.

B: Leaves: narrow lanceolate, dark green on the surface and lighter beneath.

C Fruit: Globular, light green, 4-5 cms diameter. Contains single nut.

D Nut: Single, triangular, with deep groove along the edge. Each nut contains 5 pale yellow seeds.

E: Seed: Contains 3 active glycosides:

1. Thevetin
2. Thevotoxin
3. Cerberin

Thevetin and Thevotoxin are isolated from kernels of the seed. Thevetin and Cerberin reside in the milky juice of all parts of the plant.

The other glycosides present in *Cerbera thevetia* are:

1. Nerifolin
2. Peruvoside
3. Ruvoside

Present in the milky juice from all parts of the plant.

TOXICOLOGY

The Oleander plant exerts predominantly 2 kinds of toxicities- 1. Cardiac and 2. CNS. The glycosides responsible for toxicitiy are found in plants and is composed of sugar and a non-sugar compound. The non-sugar compound has toxicological action. Yellow Oleander contains within their tissue cardenolides that are capable of exerting a positive inotropic effect on human heart.

Thevetin:

Present in about 3-4 % of the yellow oleander seed. It is 1/8 potent as oubain and its action is similar to digitalis in the heart.

Thevotoxin:

It acts similar to that of the thevetin but less toxic.

Cerberin:

Action similar to strychnine. Acts on CNS producing tetanoid convulsions.

Nerifolin:

More potent than thevetin.

Peruvoside and Ruvoside:

Acts on CNS producing tetanoid convulsions.

Fatal dose: 8 - 10 seeds

15 – 20 g root

Fatal period: 2 to 3 hrs upto 24 hrs.

Cardiac Toxicity:

This is due to the cardiotoxic property of glycosides in seeds. The basis for the physiological action of yellow oleander cardenolides is similar to classic digitalis glycosides that inhibit plasmalemmal Na – K ATP ase. But differences in toxicity and extra cardiac effect exist between oleander and digitalis cardenolides.

The human mortality associated with oleander seed ingestion is generally low.

Thevetia contains 3 cardiac glycosides

1.Thevetia – a

2.Thevetia – b

3.Thevotoxin

Thevetia b is identical to digitalis. Many studies have demonstrated that, like digoxin, uniform distribution throughout the body does not occur and in fact levels in heart tissue are 20 times more. Two leaves of oleander are sufficient to raise the toxic glycoside levels. Kernels are more toxic. Ingestion of one kernel can produce severe symptoms.

Electro Physiological properties of cardiac glycosides:

The conduction tissue in atria and purkinje fibres are more sensitive.

1: Effect on RMP: The RMP is progressively decreased with higher dose of cardiac glycosides. Excitation is enhanced at lower dose and depressed in toxic dose. Action potential is reduced in phase-2 and the amplitude also diminished.

2: Effect on ERP: In the atrium, ERP is decreased by vagal action and increased by direct action. In AV node and bundle of His increased by direct action. In the ventricle also, it is decreased by direct action.

3:Conduction system: A–V conduction is slowed by therapeutic doses due to reduction in the rate of phase-0 of depolarization.

ECG changes:

High doses of cardiac glycosides cause arrhythmias and following changes.

1. Decrease in amplitude or inversion of T wave
2. Increase in PR interval
3. AV block at toxic doses
4. Short QT interval – reflects abnormal shortening of systole
5. Depression of ST segment at high doses of cardiac glycosides.

Mechanism of action:

Digitalis selectively binds to the membrane associated Na–K ATPase of the myocardial fibres and inhibit their enzymes resulting in intracellular Na accumulation. Normally intracellular Ca is exchanged with extra cellular Na . This is reduced as the intracellular Na increases and indirectly intra cellular Ca increases. This enhances the inflow of

Ca through slow Ca channel during plateau phase.Increase Ca entering the cell releases more Ca from sarcoplasmic reticulum {calcium induced calcium release}. Thus positive inotropic action results.

Inhibition of NA – K ATP ase is clearly involved in the toxic action of glycosides. At high doses there is depletion of intracellular potassium. Toxicity is reversed by potassium infusion.

CVS: Produce almost every type of arrhythmias like bigemini, VPCs, VT, VF, partial or complete AV Block, severe bradycardia, APCs, AF.

Cardiac glycosides slow the heart rate by vagal and extra vagal action.

Vagal: Vagal tone is increased reflexively through Nodose ganglion and sensitization of baroreceptors, Direct vagal centre stimulation, Sensitization of SA Node to acetylcholine.

Extra vagal: A direct action on SA and AV node probably as anti adrenergic action exerted beyond the receptor level.

Vagal action is very early and is blocked by atropine. Extra vagal action occurs late.

CNS Toxicity: CTZ activation results in nausea and vomiting.

Animal Studies

The toxic effects of the yellow oleander seed kernels were evaluated on roof rats. Crushed ground seed kernels were fed with Bart in 20 – 30 % concentrations. The signs of poisoning observed were hind limb paralysis, rolling of the body along the long axis, muscular twitch, tetanic convulsions, tremors, collapse and death. They also observed decrease in RBC count, Hb %, total count, decrease in neutrophil and increase lymphocyte, decrease blood glucose and serum proteins. Increase in BUN, SGOT and LDH.

HISTOPATHOLOGY:

Inflammatory and degenerative changes in liver and kidney, severe to moderate fatty metamorphosis, congestion, nuclear degeneration, pyknosis and necrosis were major changes in liver.

Proliferation of glomerular endothelium, hypercellularity of the glomerulus, necrosis of the convoluted tubular epithelium . disappearance of nuclei and pyknosis were important changes in the renal cortex .

Post mortem findings:

1. Signs of GIT irritation
2. Congestion of various organs
3. Generalized venous engorgement
4. Subendocardial ecchymosis
5. Hemorrhage of left side of the heart
6. Petechial hemorrhages on the heart are characteristic feature
7. Congestion of stomach and duodenum

Post mortem findings of Yellow Oleander poisoning with jaundice and renal failure :

1. Renal tubular necrosis with obliteration of the lumen of the tubules and vacuolated areas with glomerular spaces.
2. Liver – focal necrosis around central vein and patchy hemorrhagic necrosis.
3. Sinusoidal dilatation
4. Congestion of GI organs
5. Petechial hemorrhages in heart

CLINICAL MANIFESTATIONS

Symptomatolgy:

1. Vomiting and Diarrhea
2. Burning pain in the mouth
3. Dryness of throat
4. Tingling and Numbness of tongue
5. Headache
6. Giddiness, Dilated pupils
7. Loss of muscle power
8. Fainting
9. Rapid weak irregular pulse
10. Hypotension
11. Heart block, collapse and death

Causes of death in yellow oleander poisoning:

Peripheral circulatory failure

1. Cardiogenic shock
2. Heart failure

Diagnosis:

1. ECG recordings: Done in every patient. 12 lead ECG immediately after admission, then 12 and 24 hours later. In patients with serious arrhythmias- continuous ECG monitoring in ICU.

2. Blood for serum electrolytes.

3. Identification of the poison– when the extract of Yellow Oleander seed and the plants are boiled with HCL, turns into a deep blue colour. If vomitus gives this colour oleander poisoning is confirmed.

4. RIA using antibodies of different specificity towards the cardiac glycosides is used to confirm the poisoning due to yellow oleander leaves.

5. Competitive immuno assay method permits rapid screening of specimens suspected to contain cardiac glycosides. This is also useful in confirming the presence of glycosides in serum of patients.

ECG changes:

I: DISTURBANCE OF IMPULSE FORMATION

1. Sinus rhythm- a. normal rhythm

b. Sinus tachycardia

c. Sinus bradycardia

2. AV nodal rhythm -- a. AV nodal extra systole

b. AV nodal tachycardia

c. Idionodal tachycardia

II: DISTURBANCE OF IMPULSE CONDUCTION:

a. SA block and sinus arrest

b. AV block

III: SECONDARY RHYTHM DISORDERS:

1. AV dissociation.

CARDIAC DYSRRYTHMIAS IN OLEANDER POISONING

1. Sinus Rhythm:

It is usually associated with normal intraventricular conduction. It is reflected by the sequential inscription of P-QRS-T complex. It has Normal P waves with rate 60 to 100 per/min and PR is usually between 80-120 ms.

2.Sinus Tachycardia:

It is defined as HR >100 bpm. In adults, during ST, the sinus node exhibits a discharge frequency between 100-160 per min, may be higher with extreme exertion. Generally has a gradual onset and termination. P-P interval vary slightly form cycle to cycle .P wave has a Normal contour but

may develop a larger amplitude. They appear before each QRS complex with a stable P-P interval. Accelerated phase 4 diastolic depolarization of sinus nodal cells are generally responsible for sinus Tachycardia. It is characterized by Normal P-QRS-T complex.

3. Sinus Bradycardia:

It is defined as heart rate < 60bpm. P waves with Normal contour occur before every QRS with constant P-P interval. It may result from excessive vagal or less sympathetic tone as well as autonomic change in sinus node.

4. SA Block (Sinoatrial exit block):

This arrhythmia is indicated in ECG by the absence of normally expected P wave. Produces a pause, the duration of which is a multiple of basic P-P interval .SA block is due to conduction disruptions of an impulse formed within SA node , which fails to depolarize atria. SA block cannot be recognized in ECG because SA nodal discharge is not recorded.

5. Type I second degree SA block:

The P-P interval progressively shortens prior to the pause. The duration of the pause is less than the P-P cycles.

6. Type II second degree SA block:

An interval without P waves that equal approximately two, three, four times the normal P-P cycle. The digitalis effect on SA node may produce Type II SA exit block.

7. AV block:

First degree – delay in conduction

Second degree – Intermittent interruption of conduction

Third degree – complete interruption of conduction

a) First degree AV block:

PR >0.2 Sec.

It may result from conduction delay in AV node or purkinje system or both.

If QRS is of normal contour and duration – AV delay.

If QRS slows Bundle branch block pattern – Purkinje system delay.

b) Second degree AV block:

Interruption of AV conduction

P wave is followed by QRS complex and ventricular beat.

i) Type I Mobitz: It is characterized by progressive lengthening of the conduction time until impulse is not conducted.

ii) Type II Mobitz: Denotes occasional or repetitive sudden block of conduction of an impulse with out prior measurable lengthening of conduction time.

iii) Third degree AV block:

Complete AV block occurs when an electrical activity does not conduct to the ventricle, therefore atria and ventricle are controlled by independent pacemaker. Atrial pacemaker may be sinus, ectopic or may result from an AV junctional focus occurring above the block with retrograde atrial conduction.The ventricular focus is usually located below the region of the block, which may be above or below HIS bundle bifurcation. The ventricular rate in complete block is less than 40 bpm but may be faster in congenital complete AV block. The ventricular rhythm is usually regular, may vary owing to VPCS, a shift in pacemaker site, an irregularly discharging pacemaker focus or autonomic influence.

Complete AV block may result from the block at the level of AV node (usually congenital) with in the Bundle of HIS. The block is distal to AV node in the purkinje system (usually acquired).

Acquired compete AV block occurs most commonly distal to the bundle of HIS owing to the Trifascicular conduction disturbance. Each P

wave is followed by a His deflection and ventricular complexes are not preceded by a His deflection. QRS complex is abnormal and ventricular rate is usually < 40 bpm.

8. AV dissociation:

Dissociated or independent beating of the atria and ventricles is called AV dissociation. It is never a primary disturbance of rhythm but a symptom of an underlying rhythm produced by following mechanisms:

a) Sinus Bradycardia that follows the escape of an AV junctional rhythm which does not capture the atria retrogradely.

b) A ventricular premature beat with out retrograde atrial capture produces complete AV dissociation.

c) Complete AV block with a ventricular escape rhythm.

d) Combination of these.

Manifestation of AV blocks are the basic disturbances producing AV dissociation. The atria in all these beat act independently from the ventricle, under the control of the sinus node, ectopic atrial or AV junctional pacemakers.

9) Incomplete AV dissociation:

Both QRS and P appear regularly spaced with out a fixed temporal relationship to each other.

a) AV junctional Extrasystole: It is an expression of an impulse arising prematurely in the AV node. If the AV nodal extrasystole occurs with retrograde spread to the atria, the sinus node is usually discharged prematurely and pause is incomplete. If without retrograde spread, there is no interference of sinus discharge and compensatory pause is complete.

b) AV nodal Escape beat: It is characterized by the late inscriptions of an AV nodal beat. The long pauses resulting in sinus Bradycardia are terminated by the AV nodal escape beats. The QRS complex is identical in contour to those of the conducted sinus beat. The near synchronous P waves of sinus rhythm are dissociated from the QRS complex and occur just before the escape beat with shorter P-P interval.

c) Idionodal Tachycardia: It is an expression of an accelerated inherent idionodal rhythm. It is also called as non-paroxysmal AV nodal tachycardia. The ECG shows normal complex, configuration and bear no relationship to P waves.

Incompete AV dissociation – A QRS complex of supraventricular contour occurs early and is preceded by a P wave at a P-R interval >120 milliseconds within a conductable range.

10) A-V nodal or junctional rhythms:

a)A-V junctional extra systole

b)A-V junctional escape beat

c)Idionodal rythms

11) VPC:

Due to premature discharge of ventricular ectopic focus. Ventricular extrasystole is premature and arises in the diastolic period of the preceeding sinus beat.The QRS complex is bizarre, widened and slurred as the discharge arise in an ectopic focus and the course of depolarization is abnormal.When the QRS complex is dominantly upright, the ST segment is depressed and T wave inverted. When the QRS is dominantly downward, the ST segment is elevated due to abnormal depolarization. The coupling interval is constant (the interval between the ectopic beat and preceding sinus beat) is constant for all extra systole. The pause follows the

extrasystole. The compensatory pause is complete. The sum of pre and post ectopic interval is exactly equal to the sum of the consecutive sinus interval.

Rule of Bigemini– Ventricular extra systole tend to follow long R– R intervals. A long cycle precipitates an ensuring ventricular extra systole. The compensatory pause of the extra systole, which tends to precipitates the further extra systole. This process results in the Bigeminal rhythm. It is common in Digitalis intoxication.

12) Idioventricular Tachycardia (Accelerated ventricular rhythm):

The inherent rate of AV nodal pacemaker– Idionodal rhythm is enhanced and rate exceeds the sinus rate. AV nodal rhythm is accelerated. This is known as Idionodal Tachycardia and inherent Idioventricular rhythm results in Idioventricular Tachycardia. Characteristics of Idioventricular Tachycardia:

1.Evidence of Bizarre QRS.

2. Relatively rapid Idioventricular rate 70-80 bpm.

3. AV dissociation as two rhythm occurs.

14)VF :

Chaotic uncoordinated ventricular depolarization may occur in glycoside toxicity.

15)Sinus arrest or Sinus pause:

It is recognized by a pause in the sinus rhythm. The P-P interval delimiting the pause does not equal a multiple of the basic P-P interval. Sinus arrest is thought to be due to a slowing or cessation of spontaneous sinus nodal automaticity. Therefore recognition of the disorder of impulse formation from SA exit blocks in patients, with sinus arrhythmia may be quite difficult.

16) Wandering Pacemaker:

This variant of sinus arrhythmia involves the passive transfer of the dominant pacemaker focus from the SA node to the latent pacemaker that have the next higher degree of automaticity located in other atrial sites or in AV junctional tissue A cyclical increase in P-P interval and P-R interval gradually shorten and may become less than 120 ms. Changes in P contour, which became negative in L1 or L2 or is lost in QRS .Wandering Pacemaker is a normal phenomenon due to augmented vagal tone.

Nerium Odorum

It is an attractive hardy shrub that thrives in tropical and sub tropical regions. Widely grown in rural areas in India, All parts of the plant are poisonous. Flowers are usually fragrant and are borne in terminal clusters. They are in white, pink, dark red 2.5 cm to 5 cm wide and have five petals. Leaves are narrow and lanceolate shaped leathery, dark green on surface and lighter beneath and 10–25 cm long. Seed pod is slim, cylindrical, turns brown, dries, and splits, release small seeds tipped with brown hair.

Active principles:

1. Nerin
2. Nerioderin
3. Karabin

Nerin:

It is similar to digitalis in action. It causes all type of arrythmias and death from cardiac failure.

Nerioderin:

It has picrotoxin like effect of muscular twitching and tetanic spasms. It is more powerful than those of strychnine.

Karabin :

Acts as digitalis and also acts on spinal cord.

Fatal dose: 15 g of root

Fatal period: 24 – 36 hrs.

The active principles of white oleander contains within their tissue cardenolides that are capable of exerting positive inotropic effect on heart. Mechanism of action is similar to digitalis.

Symptoms and signs of Nerium Odorum poison:

1. Difficulty in swallowing and articulation
2. Abdominal pain, vomiting and sometimes diarrhoea.
3. Profuse frothy salivation.
4. Pulse: early-slow
   late-rapid and weak
5. Hypotension
6. Hurried respiration
7. Dilated pupil
8. Muscular twitching, tetanic spasms, lock jaw

9. Drowsiness and coma

10. Contact dermatitis

Diagnosis :

1. ECG recordings: done in every patient .12 lead ECG immediately after admission, then 12 and 24 hours later.

2. In patients with serious arrhythmias: continuous ECG monitoring in Intensive care unit.

3. Blood for serum electrolytes.

4. RIA using antibodies of different specificity towards the cardiac glycosides is used to confirm the poisoning due to White Oleander leaves.

5. Competitive immuno assay method permits rapid screening of specimens suspected to contain cardiac glycosides. This is also useful in confirming the presence of glycosides in serum.

ECG changes :

Various degrees of conduction defects like sinus bradycardia, first degree A-V block, second degree, complete heart block, A-V dissociation, ventricular premature complexes and bundle branch block.

Post mortem findings:

1. Congestion of various organs
2. Petechial hemorrhages on heart are characteristic
3. As toxins of *Nerium odorum* resist heat it can be identified even from the burnt remains of the dead body

Cause of death: Cardiac failure, Respiratory paralysis, coma and death.

DIFFERENCES BETWEEN YELLOW AND WHITE OLEANDER.

YELLOW OLEANDER	WHITE OLEANDER
1. Flowers- yellow	**1.White**
2. Seed- large and triangular	**2.Small and cylindrical**
3. All parts poisonous	**3.All parts are poisonous**
4. Active principles: **Thevetin– powerful cardiotoxin** **Thevo toxin– less toxic** **Cerberin– acts on spinal cord**	**4.Active principles:** **Nerin– cardiotoxic** **Nerioderin– acts on spinal cord** **Karabin – both**
5. Fatal dose – 15- 20 g root **8-10 seeds**	**5. Fatal dose- 15 g root**
6. Fatal period– 2-3 hrs to 24 hrs	**6. Fatal period-24–36 hrs**
7. Cause of death –Peripheral circulatory failure, cardiogenic shockHeart failure	**7. Cause of death-Cardiac failure Respiratory paralysis**
8. Post mortem findings: Resists putrefaction and so can be identified even in putrefied body	**8. Post mortem findings: Resists heat and so can be identified even from ash.**

MATERIALS AND METHODS:

100 patients of Yellow Oleander poisoning have been selected randomly in intensive medical care unit in our hospital from April 2004 to August 2004.

1. Detailed history was taken:

    a. Regarding the number of seeds consumed
    b. Reason for consumption
    c. Mode of intake– swallowed or chewed or grounded or paste form.
    d. Time of consumption– whether taken with empty stomach or after food, if taken after food {how many hours}.
    e. Whether yellow oleander seed is taken alone or with other poisons like organophosphorus, cow dung powder, alcohol or coffee.
    f. Symptoms at the time of presentation
    g. Time between ingestion and admission
    h. History of first aid

2. Complete clinical examination:

G/E: Pulse Rate, Rhythm; Blood pressure; Respiratory Rate; Single Breath Count.

CVS: JVP(Cannon a waves); Varying intensity of first heart sound; regularity of heart sounds.

CNS: GCS, Pupil size& reactions; Neuromuscular weakness; Respiratory muscle paralysis.

3.ECG:

Recordings were taken in 12 lead ECG immediately after admission. Patients with arrhythmias were put on continuous ECG monitoring and recordings were taken in lead 2 long strip. Then ECG was repeated 12 hrs later and every 24 hrs till discharge.

RESULTS AND OBSERVATION :

TABULATION SHOWING AGE INCIDENCE IN 100 PATIENTS OF OLEANDAR SEED POISONING:

TABLE I:

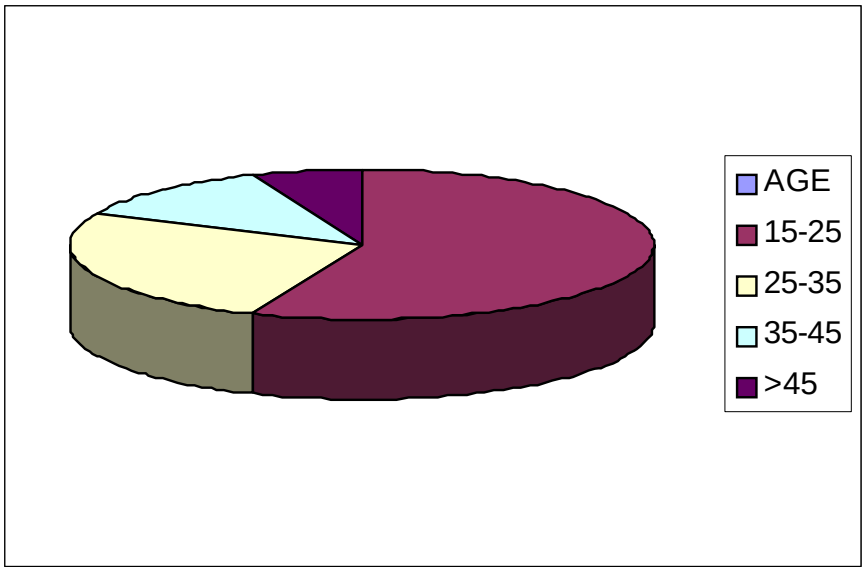
Age	No of patients	Percentage
15-25	56	56%
25-35	26	26%
35-45	12	12%
>45	6	6%

Discussion:

The vulnerable age group for oleander seed poisoning was 15-35 years, which contributed to 82 % of the study population. Only 6% of the patients were >45 years.12% of the patients were among the age group of 35–45 years.

PIE DIAGRAM SHOWING AGE INCIDENCE OF

OLEANDER SEED POISONING IN 100 PATIENTS



Discussion:

Out of the 100 selected patients around 56 % of the patients selected were in the range of 15– 25 yrs. 26 % of the patients in the age range of 25– 35 yrs. 12 % of patients in 35– 45 yrs and 6 % in the age group more than 45 yrs. Thus according to the study the incidence of the poisoning is higher in the age group of 15– 25 yrs.

SEX INCIDENCE AMONG 100 PATIENTS OF OLEANDER SEED POISONING

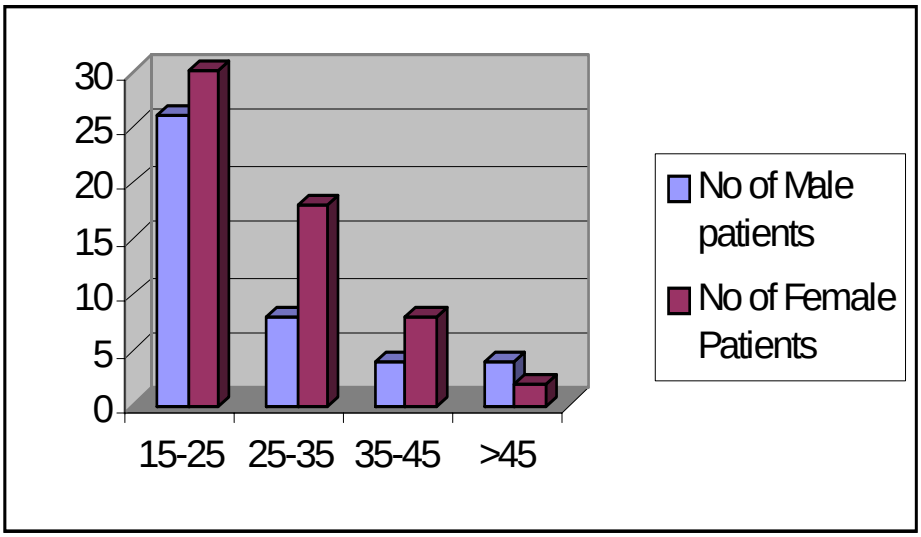
TABLE II:

Age	No of Male patients:	Male(%):	No of Female patients:	Female(%):
15-25	26	26%	30	30%
25-35	8	8%	18	18%
35-45	4	4%	8	8%
>45	4	4%	2	2%

Discussion :

The incidence of poisoning in males was 42 %. 26 % in the age group between15 – 25 yrs constituting the major portion. Among females the incidence of poisoning was 58% with 30% in the age group between 15–25 yrs and 18 % in 25 – 35 yrs. Thus the incidence of poisoning is higher in females than males.

BAR DIAGRAM SHOWING SEX INCIDENCE AMONG 100 PATIENTS OF OLEANDER SEED POISONING:



Discussion:

The incidence of oleander seed poisoning was higher among female patients. Majority of females were among 15–35 years age group. 42 % of males had consumed oleander seed. Even among males 34 % of them were among 15–35 years age group.

TABULATION SHOWING RELATIONSHIP BETWEEN AGE GROUP & MORTALITY AMONG 100 PATIENTS OF OLEANDER SEED POISONING.

TABLE III:

Age	No of Deaths	Percentage
15-25	2	40%
25-35	2	40%
35-45	0	0%
>45	1	20%

Discussion :

Total number of deaths in our study population was 5 % .4 % among the age group of 15 and 35 yrs.1 % in the age group of > 45 yrs. The commonest cause of death being bradyarrythmias. 2 % of deaths was due to second degree A-V block. Sudden cardiac death was the cause in another 2% .In 1% of the population mortality was due to complete heart block.

TABULATION SHOWING RELATIONSHIP BETWEEN MORTALITY &GENDER IN 100 PATIENTS OF OLEANDER SEED POISONING:

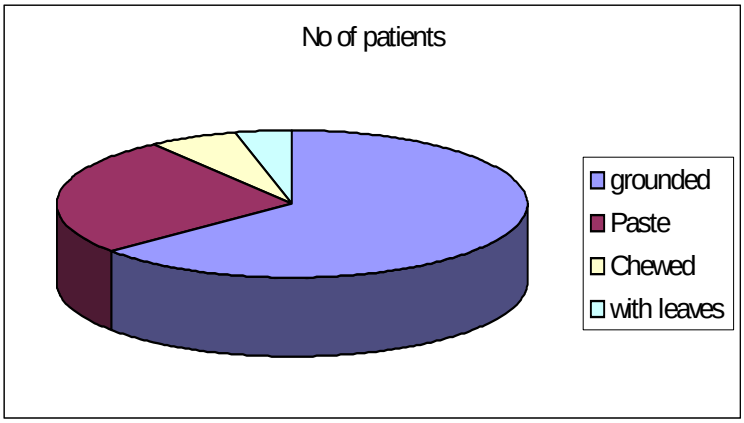
TABLE IV :

Total deaths (%)	Males (%)	Females (%)
5 (5%)	3 (3%)	2 (2%)

Discussion :

Total number of deaths in the selected study group of 100 patients was 5 % . Males contributed to 3 % of mortality. 2 % of mortality was seen among females. According to the study mortality is more in males, though the incidence of poisoning is more in female patients. This may be attributed to the number of seeds taken, the mode of consumption, time window for hospitalisation. Though the incidence of poisoning was more common in females, the mortality is more common in males. This may be related more to the mode of consumption of the seed rather than the sex difference.

PIE DIAGRAM SHOWING THE RELATIVE INCIDENCE OF THE FORM OF OLEANDER SEED CONSUMED IN 100 PATIENTS.



Discussion :

The mode of consumption in majority of patients in our study population was in grounded form, around 64%. 26 % of patients have taken it in paste form. The toxin availability in these forms are more ,hence mortality is more when the seed is taken in these forms .other modes of consumption seen in the study group are chewed seeds and seeds along with leaves.

TABULATION SHOWING THE VARIOUS FORMS OF CONSUMPTION OF OLEANDER SEED IN 100 PATIENTS:

TABLE V:

Forms of consumption	Number of patients { % }
Grounded	64{ 64% }
Paste	26 { 26%}
Chewed	6 { 6 % }
Seeds with leaves	4 { 4% }

Discussion :

Out of the 100 patients 64 patients took the seed in grounded form, 26 patients took it in the paste form , 6 patients in chewed form and 4 patients have taken seeds with leaves. In the grounded and paste form the alkaloid availability is more and hence the cardiac toxicity .

TABULATION SHOWING THE RELATIONSHIP BETWEEN SOCIO ECONOMIC STATUS & OLEANDER SEED POISONING IN 100 PATIENTS.

TABLE VI :

Socio economic class	No. of patients	Percentage
Class I	**--**	**--**
Class II	**--**	**--**
Class III	**--**	**--**
Class IV	**31**	**31%**
Class V	**69**	**69%**

Discussion :

Out of the 100 patients 31 % were from class IV and 69 % were from class V . The incidence of poisoning according to the study was more common in patents of lower socio economic status.

TABULATION SHOWING TIME WINDOW BETWEEN CONSUMPTION AND HOSPITALISATION IN 100 PATIENTS OF OLEANDER SEED POISONING:

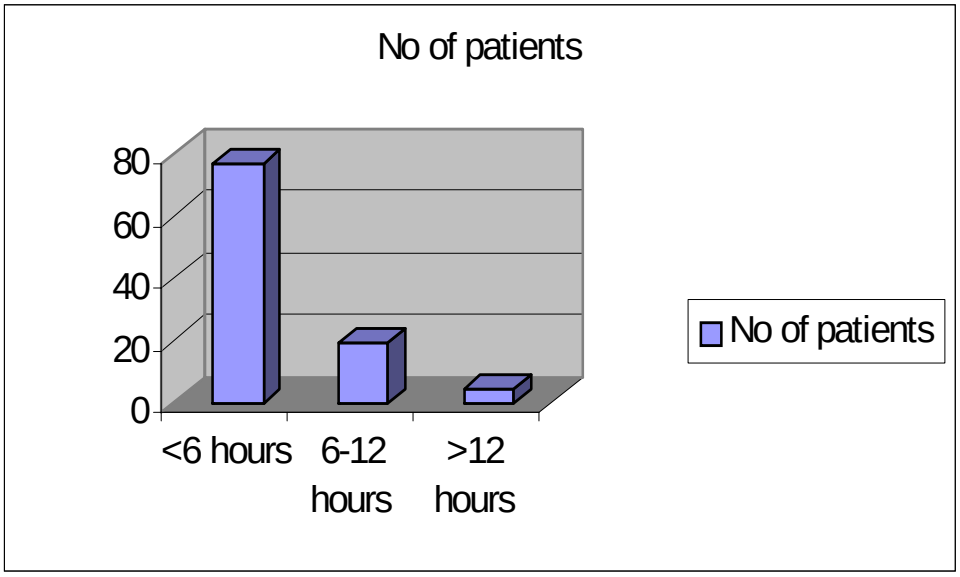
TABLE VII:

Time interval	No. of patients	Percentage
< 6 hrs	77	77%
6 – 12 hrs	19	19%
> 12 hrs	4	4%

Discussion :

Out of the total 100patients 77 % of patients presented with in 6 hrs of consumption.19% patients presented within 6– 12 hrs and only 4% of patients presented beyond 12 hours. Most of the patients presented earlier (< 6hrs), hence the low level of mortality observed in our study.

BAR DIAGRAM SHOWING TIME WINDOW BETWEEN CONSUMPTION AND HOSPITALISATION IN 100 PATIENTS OF OLEANDER SEED POISONING



Discussion :

In majoritty of the study population the time window was < 6 hours.Delayed presentation was seen only in 4 % of patients.This may be because of the awareness of the study group.this can even attribute to the low incidence of mortality.

TABULATION SHOWING THE AMOUNT OF OLEANDER SEEDS CONSUMED IN 100 PATIENTS:

TABLE VIII :

Number of seeds	Number of patients	Percentage
1 – 3	**13**	**13 %**
4 - 6	**67**	**67 %**
7 – 9	**14**	**14 %**
10 – 12	**6**	**6 %**

Discussion :

In our study most patients consumed around 4 – 6 seeds constituting 67 % .Mortality was not seen when patients consumed < 3 seeds . Mortality appears high when patient consumed > 5 seeds .There appears to be a direct relationship between the number of seeds consumed , ECG abnormality and mortality.

TABULATION SHOWING THE INCIDENCE OF ECG ABNORMALITIES IN 100 PATIENTS OF OLEANDER SEED POISONING

TABLE IX:

Total patients	Abnormal Ecg	%	Normal Ecg	%
100	78	78 %	22	22 %

Discussion :

78 % of the patients had ECG abnormalities and remaining 22 had normal ECG . The abscence of Ecg changes in 22 % of patients can be attributed to the less no : of seeds consumed ,effective first aid and partly to mode of consumption .78 % of patients who had Ecg changes had mostly consumed > 3 seeds ,in grounded or pasted form which has a higher alkaloid content.

TABULATION SHOWING THE RELATIONSHIP BETWEEN AGE & ABNORMAL ECG IN 100 PATIENTS OF OLEANDER SEED POISONING.

TABLE X:

Age group (yrs)	Number of patients	Percentage
15 – 25	46	60 %
25 – 35	13	17 %
35-45	11	14 %
>45	8	9 %

Discussion :

The number of patients with ECG abnormality was 46 in the age group of 15 – 25 yrs constituting around 60 % . 17 % of patients had ECG abnormality in 25 – 35 yrs age group. In 35 – 45 yrs 14 % and in > 45 yrs 9 % of patients had ECG abnormality.

TABULATION SHOWING THE RELATIONSHIP BETWEEN GENDER & ABNORMAL ECG IN 100 PATIENTS OF OLEANDER SEED POISONING.

TABLE XI :

Patients with abnormal ECG	Male (%)	Female (%)
78	**33 (42.3 %)**	**45 (57.7 %)**

Discussion :

In our study 78 patients had Ecg abnormality. 33 male patients had Ecg abnormality,which was around 42.3 % and 45 female patients had abnormal Ecg ie 57.7 % . Thus according to the study ECG abnormality was more common in females than males .

TABULATION SHOWING THE NO: OFOLEANDER SEEDS CONSUMED & ABNORMAL ECG IN 100 PATIENTS .

TABLE XII:

Number of seeds consumed	Patients with abnormal Ecg	Percentage
< 3	6	46.2 %
>3	72	82.8 %

Discussion :

Out of the 13 patients who had consumed < 3 seeds 6 patients had ECG abnormality . 72 Patients who had consumed more than 3 seeds had ECG abnormality . Thus around 83 % of patients who had consumed > 3 seeds had abnormal ECG. ECG abnormality was more common if > 3 seeds are consumed.

TABULATION SHOWING THE VARIOUS FORMS OF ECG ABNORMALITY IN 100 PATIENTS OF OLEANDER SEED POISONING

TABLE XII:

Forms of Ecg abnormality	No. of patients with abnormal ECG	%
Normal sinus rhythm	**22**	**22 %**
Brady arrythmia	**54**	**54 %**
Tachyarrythmia	**24**	**24 %**

Discussion :

Out of the 78 patients with ECG abnormality 54 had bradyarrythmia and 24 had tachy arrythmia. According to the study bradyarrythmias were more common than tachy arrythmias. Commonest form of arrythmias are sinus bradycardia followed by first degree A-V block. Rarer forms include type 2 mobitz and complete A-V block . sinus tachycardia was the most common tachyarrythmia observed.

TABULATION SHOWING THE RELATIONSHIP BETWEEN FORMS OF ECG ABNORMALITY & MORTALITY IN 100 PATIENTS OF OLEANDER SEED POISONING

TABLE XIV:

Ecg abnormality	No. of deaths	Percentage
Second degree A – V block	2	40 %
Complete heart block	1	20 %
Sudden cardiac death	2	40 %

Discussion :

Out of the 5 deaths observed in our study ,2 patients died of second degree A-V block , 1 patient died of complete heart block and the other 2 died of sudden cardiac death .

TABULATION SHOWING THE TIME OF DISAPPEARANCE OF ABNORMAL ECG IN 100 PATIENTS OF OLEANDER SEED POISONING.

TABLE XV:

Time of disappearance	No: of patients	%
Reverted with in 24 hrs	29	37.17
24 – 48 hrs	27	34.61
48 – 72 hrs	13	16 .66
72 – 96 hrs	6	07.69
Upto 1 week	3	03.84

Discussion :

Out of the 78 patients in the study who had ECG abnormality ,changes reverted in 48 hrs in 56 patients , in 13 patients it reverted in 72 hrs and in 6 patients in 96 hrs.ECG abnormality persisted in 3 patients till 1 week .

CONCLUSION

1. Common age group of poisoning in our study is 15 – 25 yrs .
2. Study reveals a little higher incidence of poisoning in females.
3. yellow oleander is consumed commonly.
4. Oleander seed is the most common part of the plant that is consumed.
5. Mortality is related to time window, no: of seeds consumed ,mode of consumption and cardiotoxicity.
6. Most common mode of consumption was in the grounded form
7. In our study mortality is seen more commonly if the consumption of seed was > 3.
8. ECG abnormality is seen in 78 % of the patients.

7.There is a marginal increase in the ECG abnormality in females

8.ECG abnormality can occur as early as 2 hrs and as late as 18hrs.

9.Commonest arrythmia found in our study is sinus bradycardia.

10.Varying arrythmias observed in the same patient mandates continuous ECG monitoring.

11.ECG abnormality lasted for 4 days in majority of patients which emphasises that monitoring should be done for a minimum of 4 days.

12.Prognosis is good if first aid is given at the earliest & time window < 6 hrs.

PROFORMA

Name

Age

Sex

Occupation

Literacy

IP no :

Ward : Unit:

Date of admission : Date of discharge:

Date and time of poisoning;

Date and time of admission

Time interval between consumption of poison and admission;

Details of poisoning :

Number of seeds with or without leaves

Quantity

Form - grounded , paste , or chewed

With food or empty stomach

With alcohol

Before admission – Vomiting yes / no

First aid

After admission

GIT symptoms

Burning sensation in mouth

Tingling sensation in tongue

Dryness of throat

Vomiting-Diarrhoea

Cardiovascular system:

Syncope

Palpitation

Dyspnoea

Central nervous system

Blurring of vision

Headache

Altered sensorium

Tetanic convulsions

Coma

Renal system

Oliguria

Clinical examination :

- Temperature
- Pulse rate
- Blood pressure
- Respiratory rate
- Pupils
- CVS / RS / ABD / CNS

Investigations:

Urine

- Albumin
- Sugar
- Deposit

Blood

- Urea
- Sugar
- Sr. creatinine
- Sr. electrolytes

ECG

Autopsy

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MASTER CHART

S.no	IP.no	Name	Age	Sex	SE Status	No. of seeds	Form of conumption	Time Window(hrs)	Ecg abnormalities	Outcome
1.	776703	Vadivu	27	F	IV	2	Grounded	<6	SB	---
2.	776761	Prabhu	24	M	IV	5	Grounded	<6	SB	---
3.	776800	Vani	17	F	IV	4	Grounded	<6	ST	---
4.	776812	Mahalakshmi	17	F	V	7	Paste	6-12	SB	---
5.	777004	Santharam	25	M	IV	3	Grounded	6-12	SB	---
6.	777005	Shanthi	22	F	V	5	Grounded	<6	ST	---
7.	777116	Srinivasan	21	M	IV	4	Grounded	<6	ST	---
8.	777295	Indu	19	F	IV	6	Grounded	<6	AVB	---
9.	777337	Gopal	23	M	V	1	Paste	<6	SB	---
10.	777339	Palani	25	M	V	5	Chewed	<6	SB	---
11.	777556	Kousalya	24	F	V	4	Paste	<6	SB	---
12.	777625	Deepa	19	F	V	6	Paste	6-12	ST	---
13.	777626	Mahendiran	26	M	IV	4	Grounded	6-12	AVB	---
14.	777780	Anbu	25	F	IV	2	Paste	6-12	SB	---
15.	777809	Anandhi	18	F	IV	5	Paste	6-12	SB	---
16.	777870	Lourdhmary	36	F	V	6	Grounded	>12	SB	---
17.	777915	Naresh	25	M	V	4	Grounded	<6	SB	---
18.	778004	Sekar	24	M	V	3	Grounded	<6	SB	---
19.	778121	Rajalakshmi	23	F	IV	5	Grounded	<6	ST	---
20.	778147	Victoria	35	F	IV	10	Grounded	6-12	AVB	---
21.	777780	Murugan	35	M	IV	6	Grounded	<6	SB	---
22.	778351	Dhanam	21	F	V	4	Paste	<6	SB	---
23.	778400	Saroja	20	F	V	1	Grounded	<6	SB	---
24.	778576	Siva	21	M	V	5	Grounded	<6	SB	---
25.	778874	Kothandam*	60	M	IV	7	Grounded	6-12	SCD	Death

26.	779172	Prema	20	F	V	4	Grounded	6-12	AVB	---
27.	779204	Sundari	21	F	V	2	Grounded	>12	ST	---
28.	779521	Padmanabhan	18	M	IV	6	Grounded	<6	SB	---
29	779523	Laxmi	17	F	V	2	Paste	<6	AVB	---
30.	779568	Rangasamy	25	M	V	5	Grounded	<6	SB	---
31.	779692	MariaSelvi	28	F	V	6	Grounded	<6	SB	---
32.	779807	Siva	22	M	V	4	Grounded	<6	SB	---
33.	780231	Lakshmi	25	F	IV	1	Grounded	<6	SB	---
34.	780240	Paneer	19	M	IV	8	Grounded	6-12	SB	---
35.	780248	Dhanabakiyam	22	F	IV	6	Paste	6-12	SB	---
36.	780481	Chitra	21	F	V	7	Paste	6-12	AVB	---
37.	780482	Muniyammal	33	F	V	4	Grounded	6-12	AVB	---
38.	780480	Selvarasu	26	M	V	3	Chewed	>12	SB	---
39.	780598	Senthil	22	M	IV	3	Grounded	<6	SB	---
40.	780618	Deepa	25	F	V	6	Grounded	<6	ST	---
41.	780726	SanthaKumari*	19	F	V	5	Grounded	>12	AVB-II	Death
42.	780727	Sekar	32	M	V	6	Grounded	<6	SB	---
43.	780826	Anjalai	36	F	V	1	Grounded	<6	SB	---
44.	780965	Suryaprakash	25	M	IV	4	Grounded	6-12	SB	---
45.	780966	Kallalbai	45	M	IV	6	Grounded	<6	SB	---
46.	780979	Raghu	23	M	V	3	Grounded	6-12	AVB	---
47.	780978	Venkatesh	24	M	V	5	Grounded	6-12	SB	---
48.	780981	Kamali	16	F	V	6	Paste	<6	ST	---
49.	780984	Sarojini	17	F	IV	4	Paste	<6	SB	---
50.	781063	Sathish	19	M	V	9	Chewed	<6	AVB	---
51.	781196	Mala	23	F	V	3	Paste	6-12	SB	---

52.	781365	Amudha	21	F	IV	5	Paste	>12	SB	---
53.	781519	Appalaraju	29	M	IV	4	Grounded	<6	SB	---
54.	781796	Selvi	19	F	V	7	Grounded	<6	SB	---
55.	781748	Geetha	17	F	V	6	Paste	<6	SB	---
56.	781914	Sudhakar	23	M	IV	2	Paste	<6	SB	---
57.	782220	Ashok Kumar	22	M	V	4	Grounded	<6	AVB	---
58.	782234	Elanchezhian	19	M	V	8	Grounded	<6	AVB	---
59.	782253	Ashokan	22	M	IV	7	Chewed	6-12	SB	---
60.	782440	Shanbagam	25	F	IV	6	Grounded	6-12	AVB	---
61.	782439	Narasimhan	32	M	IV	5	Grounded	6-12	SB	---
62.	782479	Raji	23	F	V	5	Paste	6-12	SB	---
63.	782582	Marimuthu	36	M	V	4	Paste	<6	SB	---
64.	782594	Viji	24	F	IV	9	WithLeaves	<6	SB	---
65.	782797	Gandhi	25	M	IV	7	Grounded	<6	SB	---
66.	782899	Sekar	26	M	V	2	Grounded	<6	SB	---
67.	782966	Sheela	21	F	V	8	Grounded	<6	AVB	---
68.	783184	Murugan	28.	M	V	6	Grounded	<6	AVB	---
69.	783292	Manimegalai	22	F	V	4	Grounded	>12	ST	---
70.	783454	Chitra	16	F	IV	4	Grounded	6-12	SB	---
71.	783489	Komalavalli	15	F	IV	6	Grounded	6-12	AVB	---
72.	783831	Kannan	18	M	IV	3	Grounded	6-12	ST	---
73.	783924	Panner	26	M	V	3	Grounded	<6	ST	---
74.	783931	Kantha	22	F	IV	6	Grounded	<6	SB	---
75.	783934	Guru	25	M	IV	4	Chewed	<6	SB	---
76.	784002	Raja	36	M	V	5	Grounded	6-12	SB	---
77.	784021	Senthil	27	M	V	11	Paste	>12	SB	---
78.	784289	Muniyan	40	M	IV	1	Paste	<6	ST	---
79.	784299	Kannan	27	M	V	6	WithLeaves	<6	ST	---

80.	784458	Vinodh	33	M	V	8	Chewed	<6	SB	---
81.	784574	Sathya	22	F	V	4	Paste	<6	ST	---
82.	784947	Sasikala	16	F	IV	6	Paste	<6	SB	---
83.	785005	Gayathiri	20	F	V	3	Grounded	<6	SB	---
84.	785143	Selvam	29	M	V	4	Paste	6-12	AVB	---
85.	785148	Vennila	17	F	V	8	Paste	6-12	AVB	---
86.	785242	Kabibunisha	15	F	IV	4	Grounded	6-12	ST	---
87.	785250	Leena*	21	F	IV	6	Grounded	6-12	AVB-II	Death
88.	785418	Senthil	23	M	IV	5	Grounded	<6	AVB	---
89.	785629	Sankar	17	M	IV	3	Paste	>12	ST	---
90.	785680	Gomathi	19	F	V	7	Chewed	6-12	ST	---
91.	786275	Parimala	28	F	V	5	WithLeaves	6-12	SB	---
92.	786740	Mohana	17	F	V	2	Paste	<6	SB	---
93.	786834	Shanthi	22	F	IV	8	Grounded	<6	SB	---
94.	786892	Kasthuri	22	F	IV	6	Grounded	<6	SB	---
95.	787064	Latha	20	F	IV	4	Grounded	<6	ST	---
96.	787524	Shantha	15	F	V	5	Paste	<6	ST	---
97.	787545	Kabali*	29	M	V	10	Paste	6-12	SCD	-Death
98.	787561	Balamal	50	F	V	1	Grounded	6-12	ST	---
99.	787702	Radha	35	F	V	4	Grounded	<6	SB	---
100	787732	Kathirmani*	25	M	V	8	Grounded	<6	CHB	Death