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INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/13574
DOI URL: http://dx.doi.org/10.21474/IJAR01/13574

RESEARCH ARTICLE

RETROSPECTIVE STUDY OF ORGANOPHOSPHORUS POISONING PATIENTS WITH REFERENCE TO CLINICAL PRESENTATION, SERUM CHOLINESTERASE LEVEL AND OUTCOME TO TREATMENT

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Manuscript Info

Manuscript History
Received: 24 August 2021
Final Accepted: 27 September 2021
Published: October 2021

Key words:-
Organophosphoruspoisoning, Suicidal, Insecticides, Respiratoryfailure, SerumCholinesteraselevel

Abstract

The present study was conducted to analyze Organophosphorus poisoning patients with respect to serum cholinesterase level and get an idea of its correlation with various other factors to predict clinical course, severity and outcome of treatment. This was a retrospective study of 6 months duration (July 1st – December 31st 2017) conducted in General Medicine and Record & Statistics Department of tertiary care hospital. The study was approved by the Institutional Ethics Committee. All the patients of OP poisoning and patients with clinical features suggestive of OP poisoning, irrespective of age/sex were included in the study. Non-OP poisoning patients, patients with renal failure and with multiple poisoning with other drugs such as opioids, diazepam, barbiturate, etc were excluded. Out of 262 poisoning cases in this study, 46 cases were OP poisoning. Sex ratio (M:F) is 3.6:1 and 16-30 yrs age group was most commonly affected, unmarried and belonging to rural area. Suicidal being the most common manner of OP poisoning and by oral route. Family stress in case of suicidal poisoning and alcohol influence for accidental poisoning emerged to be the most common reasons. The mean SCE level is 4491 ± 4128 U/L. Most cases were found to be in the range of 1000-2000U/L (32.6%). Mortality rate in this study was only 13%, due to the close proximity of medical facilities available in our region. Vomiting was found to be the most common clinical feature, followed by giddiness and miosis. Hospital stay duration was seen to be more in patients with low level of SCE (< 2000 U/L). Duration of ventilation needed was more among the patients with low level of SCE (< 2000 U/L). But, SCE level did not show any correlation with treatment's outcome, blood pressure, respiratory rate, breathlessness, vomiting and unconsciousness. Treatment's outcome worsened with increase in gap between OP consumption and hospital admission.

Introduction:-

Poisoning,eitherhomicidalorsuicidalaresignificantcontributortomortalityandmorbidityinhumansthroughouttheworld.AccordingtoNationalPoisonInformationCentreIndia,OrganophosphorusPoisoning(OP)isamongthemostcommonmodalityofs

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uicidalpoisoning.WHOestimatedthateveryyearabout3millionpesticidepoisoningcaseswith2,20,000deathsoccur throughouttheworld.[1]Indevelopingcountries,the mortality ratecanbeas high as70 %.[2][3]

InIndia,OPpoisoningisthemostcommonpoisoning,becauseofagriculturebasedeconomy,poverty,easyaccesstohighlytoxic OPpoisons,unsafepracticesofitsuseandlackofknowledgeandprotectiveclothing.Patternofpoisoninginanyregiondependson variousfactorssuchasavailabilityofpoisonandsocio-economicstatusofpopulation.[4][5]Itisthereforeveryimporttanttodevelopawarenessandeducatefarmersforsafeapplicationp racticeofOPbasedpesticidesfor agricultural and domestic uses.

LocalexposuretoOPpoisonsshowlocalmuscarinicmanifestationwhichoccursimmediatelyandisfollowedbycomplexsystem iceffectsduetomuscarinic,nicotinicandcentralactions.Inadditiontotheabovemanifestations,somecholinesteraseinhibitors mayalsocausedelayed,oftenpermanentperipheralneuropathy.[6]Deathoccursgenerallyduetorespiratoryfailure.[7]OPpoiso nsinhibitbothacetylcholinesteraseandserumcholinesterase(SCE) activities, as theyare irreversiblecholinesteraseinhibitor.The inhibition ofcholinesteraseactivityleadstoaccumulationofacetylcholineanddisruptionofneurotransmissioninbothcentral andperipheralnervous system.

Thefatalityisoftenrelatedtoadelayindiagnosisoranimpropermanagement.[8]Currenttreatmentmainlyaimsatgastrointestina lmucosaldecontamination,followedbyadministrationofatropineandoximesasantidotetocontrolthesymptomsandreversethe effectsof theOP compounds.

Earlyrecognition,promptsupportivemeasuresandtreatmentmayimprovesurvival.InIndiansetup,owingtolimitedavailabilit yofresources,allOPpoisoningpatientsarenotmanagedinICUs.Hencethemortalityiscomparativelyhigherthandevelopedcou ntries.Itisthereforeimportantthatclinical features andcriteriato predictthecomplicationsandseverity ofpoisoningbeidentifiedatinitialexamination.Serumcholinesteraselevelsareeasiertoestimateandusuallydepressedafter OP poisoning.Itisthereforeofparamountimportancetoexaminetherelationshipofserumcholinesteraselevelandhealthoutcomeof theOPpoisonedpatienttohelpoptimaltreatmentanddetermineprognosis.Hencethisstudyhasbeenundertakentoassesstheseve rityofOPcompoundpoisoningbyestimatingserumcholinesteraselevelsandpredict prognosis,outcomeandcomplicationsusingSCE level.

ReviewofLiterature:-

PoisoningwithOrganophosphorus(OP)compoundsisaglobalproblem.OPcompoundsareusedaspesticidesandherbicidesina griculture,aswellaschemicalwarfareagents inthe formofnervegases, as inWorldWar2.[9]Duetoeasyavailability,excessiveuseofOPpesticidesnotonlydoesharmstoagriculturalproductionbutalso directlyorindirectlyaffectshealthofpeople.Becauseofeasieraccess,OPcompoundsareusedforsuicidal,homicidalpurposesan dsometimesaccidentalcasescanalsobefound.

AccordingtoFAO,since70%ofIndianruralpopulationstilldependsprimarilyonagriculture,pesticidesareroutinelyused.Thus ,IndiarankssecondinAsiainannualpesticideconsumption.[10]OutofalltheOPpoisoningandsubsequentdeathcasesdetected, majorityweredeliberateself-ingestion(suicidalmanner),particularlyinyoungandproductiveagegroup.Alsoduetoeasyavailabilityofhighlytoxicpesticide, itcanbereadilyreachedouttoduringstressduetofamilyproblem,failureinloveandexamphobia.[11]

Howeveritisthedeliberateself-poisoningthatischieflyresponsibleformostofthedeathsandtheimmensestrainthepesticidesputonhospitalservicesparticularl yinAsia.[12][13]Nevertheless,recentworkhasbeguntoemphasizeitsimportanceindevelopingcountrieslikeIndia.Accidental causeofOPpoisoningisduringitsuseinagriculturalpractices,specificallyduetolackofknowledgeofitswayofuseandlackofpro tectiveclothing.

Patternofpoisoninginaregiondependsonvarietyoffactorssuchasavailabilityofthepoisons, SEstatusofthepopulationandreligious/cultural influences.[4]

MechanismofAction:
Acetylcholineishydrolyzedbytheenzymecholinesterasetocholineandacetate.Choline isactivelytakenupbytheaxonalmembrane.Organophosphateirreversiblyinhibitstheenzymeacetylcholinesterase(AChE)foundatsynapticjunctions,redbloodcells(RBCs),andbutyrylcholinesterase(alsoknownaspseudocholinesteraseorplasmacholinesterase)intheblood,astheyareirreversiblecholinesteraseinhibitor.BlockadeofAChE leadsto the accumulationofexcessiveacetylcholine(ACh) at muscarinicreceptors(cholinergiceffectorcells),atnicotinicreceptors(skeletalneuromuscularjunctionsandautonomicganglia), and in theCNS.[6]

ThisaccumulationofexcessiveAchatreceptorswillcauseexcessivestimulationofreceptorsleadingtoimpairmentofsignaltransmissionanditsfinaltermination,resultingin paralysis.

Pseudocholinesteraseisanonspecifictypeofenzymeoccurringinthebodyinplasma,liver,intestineandwhitematter.Its main functionishydrolysisofingestedesters.Achisslowlyhydrolyzed,benzoylcholineandbutyrylcholineishydrolyzedwhichcannotbehydrolyzed byacetyl cholinesterase.[14]

Epidemiologyof OP poisoning:-

1. **CausativeFactor**

Organophosphoruscompoundsuchasmalathion,diazinon,etcareusedasinsecticides,pesticidesandherbicides.

2. *Recipients*

Mostlypeople who are -

i. Youngaged
ii. Male
iii. Illiterateandlessknowledgeacquainted
iv. Farmer byoccupation
v. Having lowsocio economicstatus
vi. Mentallyunstable

3. *Environmental*

OPpoisoningismoreprevalentinruralareas.Accidentalpoisoningiscausedduetoimproperuseof OPpesticidewhilespraying oncrops,whichiscommonduringmonthofJune,JulyandAugust.Whilesuicidalpoisoningisirrespectiveoftheseasonaluseof OP pesticide,hencevariationscanbe seen.

A. ClinicalFeatures:[6]

1. Duetooverstimulationofmuscarinicacetylcholine receptors– Meiosis,nausea,vomiting.
2. Duetooverstimulationofnicotinicacetylcholine receptors- Respiratoryfailure,tachycardia
3. Duetooverstimulationof nicotinicandmuscarinicacetylcholinereceptorsinCNS- Confusion,agitation,seizures, coma, respiratoryfailure
4. Duetooverstimulationofnicotinicacetylcholinereceptorsattheneuromuscularjunction– Muscleweakness,paralysis, fasciculations

Diagnosis:[6]

1. Basedonthehistoryof exposure.
2. PresenceofcharacteristicmuscarinicnicotinicandCNSmanifestationsofOPpoisoning.
3. Decreaseintheplasmapseudocholinesterase(PChE)andredbloodcellacetylcholinesterase(RBCAChE)activitiescanbe usedaslaboratoryevidenceofpoisoning.
4. PChE activityis asensitive indicator ofexposure.

Otherusefullaboratorystudiesincludeelectrolytes,glucose,BUN,creatinine,livertransaminases,arterialbloodgasesoroximetry,ECGmonitoring,andchestX-ray(ifpulmonaryedemaor aspirationofhydrocarbonsolvent is suspected).

B. Management:[6]

1. Likelyto bebeneficial
   - Washingthepoisonedperson andremoving contaminatedclothes.
   - Atropine,benzodiazepinestocontrolorganophosphorusinducedseizures,glycopyrroniumbromide(glycopyrrolate).
2. Unknowneffectiveness
   - Activatedcharcoal(singleormultipledose),alpha2adrenergicreceptoragonists,butyrylcholinesterasereplacementtherapy,extracorporealclearance,gastriclavage,magnesiumsulphate,milkorotherhomeremedyimmediatelyafteringestion, N-methyl-Daspartatereceptorantagonists,organophosphorushydrolases,oximes,sodiumbicarbonate.

C. Emergencyand Supportivemeasures:[6]

1. If necessarymaintainan openairwayand assistventilation.
2. Administersupplementaloxygen if necessary.
3. Treathydrocarbonpneumonitis,seizures,andcoma iftheyoccur.
4. Observeasymptomaticpatientsforatleast8to12hourstoruleoutdelayed-onsetsymptoms,especiallyafterextensiveskinexposureoringestionofahighlyfat-solubleagent.

D. SpecificDrugs andAntidotes:[6]

1. Giveatropine**,**0.5mgto2mgIVinitially,andthendoublethedoseevery5minutesuntilsignsofatropinizationarepresent(decreasedsecretionsandwheezing, increasedheartrate).
2. Pralidoxime(2-PAM)isaspecificantidotethatactstoregeneratetheenzymeactivityat all affectedsitesprior toaging.
3. Permanentinhibitionofacetylcholinesterase may occurthroughcovalent bindingbytheOPtotheenzyme.Thisisknownas“aging”anditsrateofdevelopmentisvariable anddependson thespecificOP.
4. Dimethylcompounds(e.g:dimethoate)generallyagemorequicklythandiethylagents(e.g:chlorpyrifos).
5. Antidotaltreatmentwithanoximemaydelaytheonsetofaging;earlyadministration ofoximes is thereforerecommended.**[15]**

E. Decontamination:[6]

1. **Skin:**Removeallcontaminatedclothingandwashexposedareaswithsoapandwater,includingthehairandunderthenails.Irrigateexposedeyeswithcopiouslukewarmwater or saline.
2. **Ingestion:** Ifconditionsare appropriateadministeractivatedcharcoal orally.Gastric lavage shouldbe done.

F. PrognosisOPpoisoning:

PrognosisofOPpoisoningisverypoor.Andthemortalityratecanbeashighas70%indevelopingcountrieslikeIndia.**[2][3]**Assessmentofseverityofpoisoningisveryimportanttoknow,sothatpropertreatmentcanbeemployed.Timelytreatmentisverycrucialforpatient’ssurvival,becausemajorityofpatientsreachedhospitalafteragreatdelayfollowingpoisoning.Reasonforhighmortalityrateamong OPpoisoning patientscan beattributedtopoorruralhealthcareservices and lack ofgoodreferral practices.
Inviewofthis,astudyisrequiredtoknowuseofserumcholinesteraseinanticipatingtheprognosisofOPpoisoningpatientsalongwiththepresentationofpatient,withreference topatient’sclinicalfeatures.
Thisretrospectivestudywillassesswhetherthelevelofserumcholinesteraseenzymecanbeusedtopredicttheclinicalcourseof OPpoisoning,it’sseverity,ventilatorhoursneeded,ICUstaydurationandtheoutcomeof thetreatment.

Aimsand Objective:-

Aims:

Tostudythecorrelationbetweenclinicalpresentationsof OPpoisoningpatientsandoutcomeoftreatmentwith theirserumcholinesteraselevels.

Objectives:-

PrimaryObjective-

1. Retrospectiveevaluationofclinicalcourseof OPpoisoninginpatientsadmittedtotertiarycare hospital.
2. Tostudytherelationshipbetweenserumcholinesteraselevelsandclinicalpresentationofpatient.
3. Tostudytherelationshipbetweenserumcholinesteraselevelsandtreatment’soutcome,hospitalstayanddurationofventilationgiven.
4. Tostudytherelationshipbetweentimebetweenpoisoningandhospitaladmissionversus treatment’soutcome.

SecondaryObjective-
Todeterminethefrequency,demographicdistribution,seasonalvariation,sex-wisedistribution,age-wisedistribution,typeofpoison,maritalstatus,mannerofpoisoning,routeofexposure,reasonofpoisoning,serumcholinesteraselevel,hospitalstayduration,durationofventilation,timeelapsedbetweenOPpoisoningandinitiationofFirstAidandhospitaltreatment,outcomeoftreatmentandcommonclinicalfeatures.

Material and Methods:-

G. Studydesign:
AtertiarycarehospitalbasedretrospectivestudyanalysisinOPpoisoningpatientswascarriedout.

H. Type of study:
Retrospective,ObservationalandRecord-basedstudy.

I. Studysite:
DepartmentofGeneralMedicineandDepartmentof Recordandstatistics.

J. Duration of study:
AllthepatientswhowereadmittedfortreatmentofOPpoisoninginMedicineICUandWardduring01/07/17- 31/12/17 (6months)are includedin thestudy.

K. Sample size:
Duringthestudyperiod,among262patientsadmittedinhospitalforpoisoningtreatment,46were OP poisoningcases.
TheprimarystudypopulationconsistsofpatientswithOPpoisoningthatwereadmittedinMedicineIntensive Care Unit (ICU) andwardsduringtherespective studyperiod.

L. Inclusioncriteria:

- PatientswhowereadmittedinMedicinedepartmentforOPpoisoningintiertiarycarehospital,irrespectiveofage/sex.
- Patientswith clinicalfeatures suggestive ofOPpoisoning.

M. Exclusioncriteria:

1. Non-OP poisoning patients.
2. Patientswith Renalfailure.
3. Patientswithmultiple poisoning with otherdrugs such asopioids,diazepam,barbiturate,etc.

Observationsand Results:-

During the study period i.e. from 01/07/17to31/12/17,15433caseswereadmittedintherespectiveTertiarycarehospitalandoutofwhich262werepoisoningcases.Outoftotalpoisoningcases46 caseswereofOrganophosphoruspoisoning.

Table1andfigure1describeseasonalvariationoftheincidenceofOPpoisoning,whichwashighestduringmonthofOctober(28.3%),followedbySeptember(19.6%).Theleastincidencewasduringmonth ofAugust(6.5%).

Season	No. ofpatients	Percent (%)
July	8	17.4
August	3	6.5
September	9	19.6
October	13	28.3
November	5	10.9
December	8	17.4
Total	46	100.0

Table1:**SeasonalVariations inincidencesofOP Poisoning**

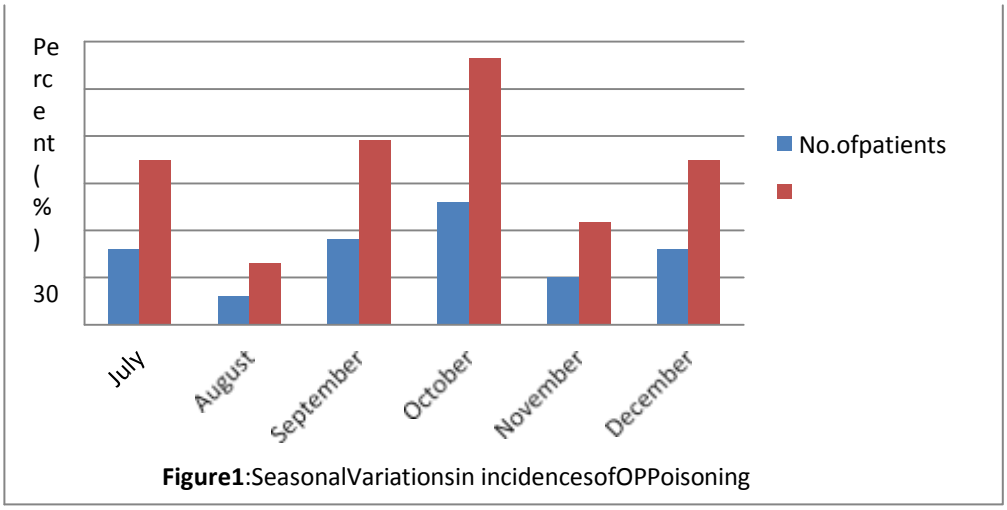


Figure1:SeasonalVariationsin incidencesofOPPoisoning

Table2andfigure2depictage-wisedistribution.Theageofpatientsvariedfrom8monthsto69years.Themeanageis29.71±12.917years.Majorityofvictimsfallin16-30yrs(67.4%),followed by 31-45yrs (21.7%).The leastbeing0-15yrs (2.2%).

Age	No. ofpatients	Percent (%)
0-15	1	2.2
16-30	31	67.4
31-45	10	21.7
46-60	3	6.5
61-75	1	2.2
Total	46	100.0

Table2:**Age-wisedistribution amongst victims of OP poisoning**

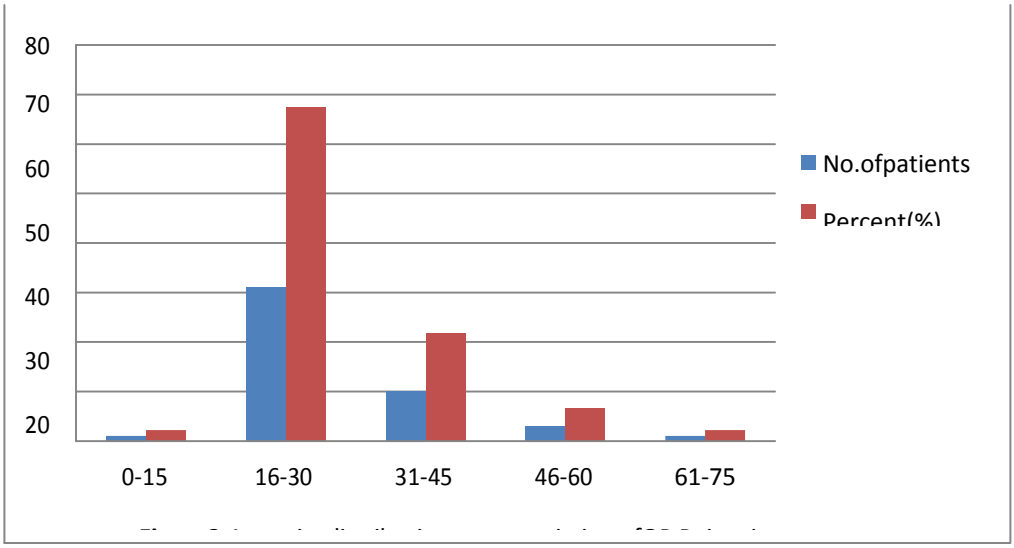


Table3andfigure3 depictsex-wise distribution. Outoftotal46 studiedcases,maleweredominantwith36cases (78.3%),femalesbeing 10cases(21.7%).

Sex	No. ofpatients	Percent (%)
Male	36	78.3
Female	10	21.7
Total	46	100.0

Table3:**Sex-wisedistribution amongstvictims of OP poisoning**

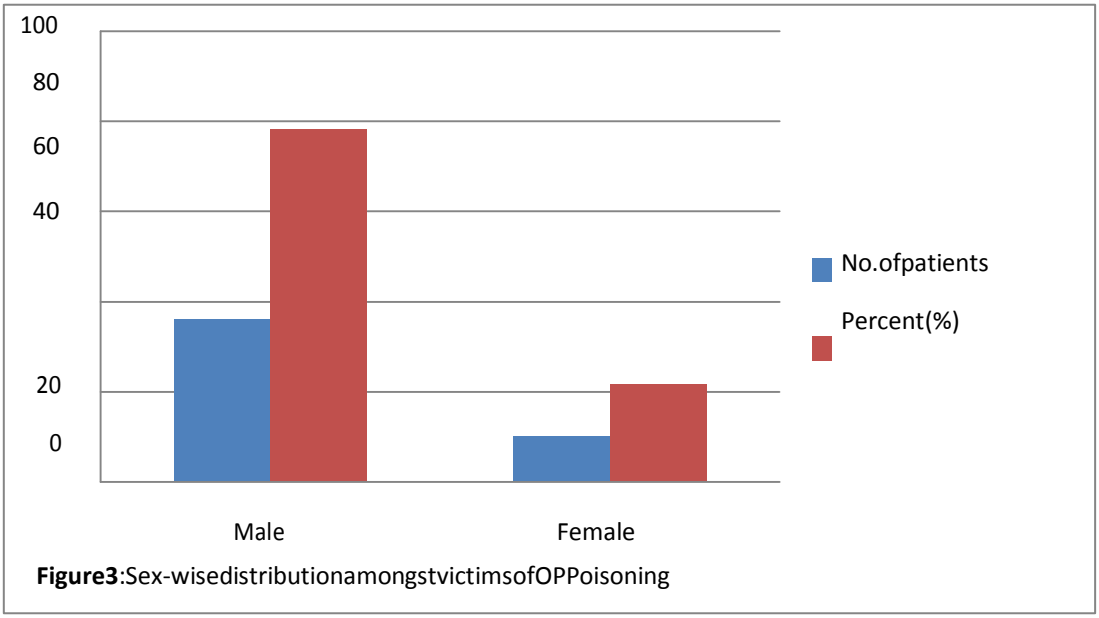


Figure3:Sex-wisedistributionamongstvictimsofOPPoisoning

Table4andfigure4describemaritalstatusofpatients.Higherincidencewasseenamongunmarried(34.8%)than married(26.1%).

Marital Status	No. ofpatients	Percent (%)
Married	12	26.1
Unmarried	16	34.8
Unknown	18	39.1
Total	46	100.0

Table4:**Maritalstatus amongst victims of OP poisoning**

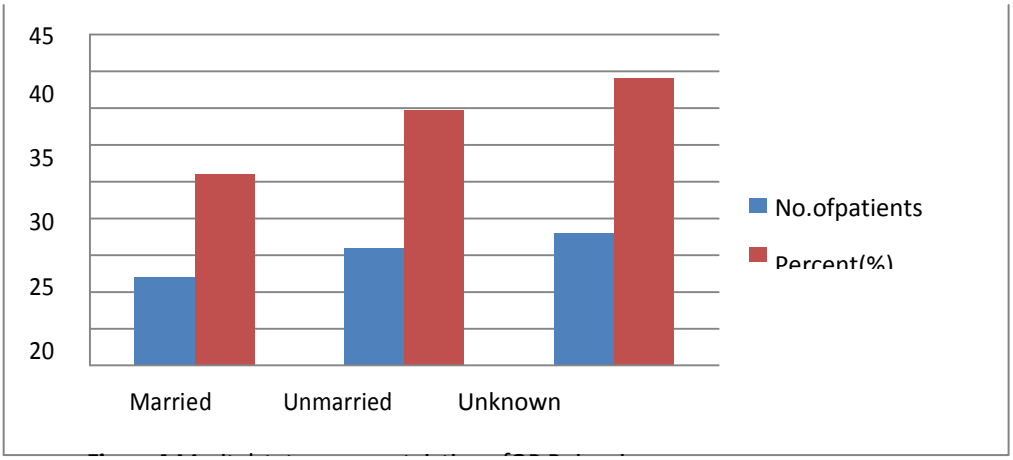


Table5andfigure5describearea-wisedistributionofOPpoisoningcases,whichwashigher inrural (91.3%)than urban(8.7%).

Area	No. ofpatients	Percent(%)
Rural	42	91.3
Urban	4	8.7
Total	46	100.0

Table5:**Area-wisedistribution amongst victims of OPPoisoning**

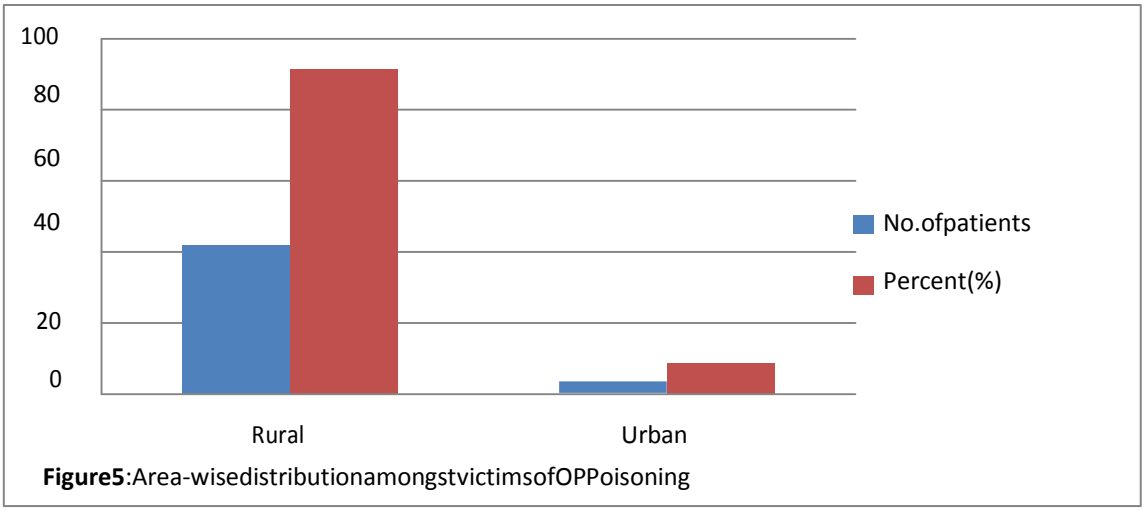


Figure5:Area-wisedistributionamongstvictimsofOPPoisoning

Table6andfigure6describetypeofOPpoisonconsumed.ThemostcommonlyconsumedOPcompoundwasDicholorvos(17.4%)andDimethoate(17.4%)andtheleastcommonbeingMalathion(2.2%)andPhorate(2.2%).

Type ofOP consumed	No. of patients	Percent (%)
Chlorpyrifos**(Hamla550)**	6	13.0
Dicholorvos**(Neon , Doom ,Nuvan)**	8	17.4
Dimethoate**(Rogor)**	8	17.4
Glyphosate	4	8.7
Malathion	1	2.2
Phorate**(Thimet)**	1	2.2
Quinalphos	2	4.3
Unknown	16	34.8
Total	46	100.0

Table6:**Different type of OP poisonconsumed**

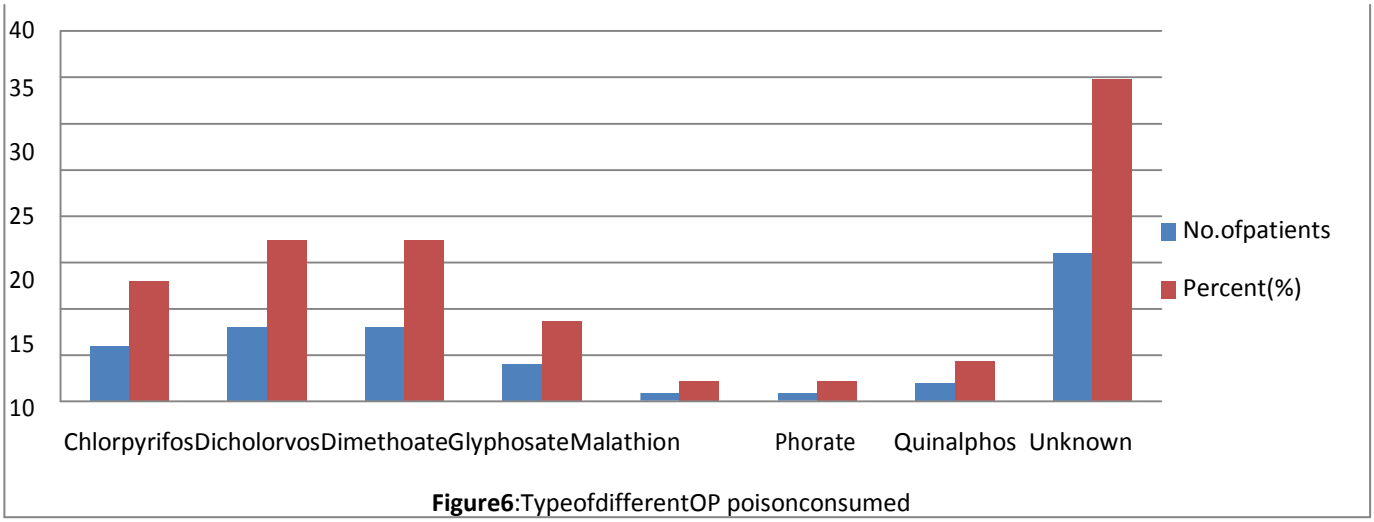


Figure6:TypeofdifferentOP poisonconsumed

Table7andfigure7describemannerofOPpoisoning,andclearlyindicatesthatOPwasusedfor suicidalpurpose (43.5%). Followedbyaccidental (28.3%),andleastbeing homicidal(2.2%).

Manner of OP poisoning	No. ofpatients	Percent (%)
Accidental	13	28.3
Homicidal	1	2.2
Suicidal	20	43.5
Unknown	12	26.1
Total	46	100.0

Table7:**Manner of OP poisoning**

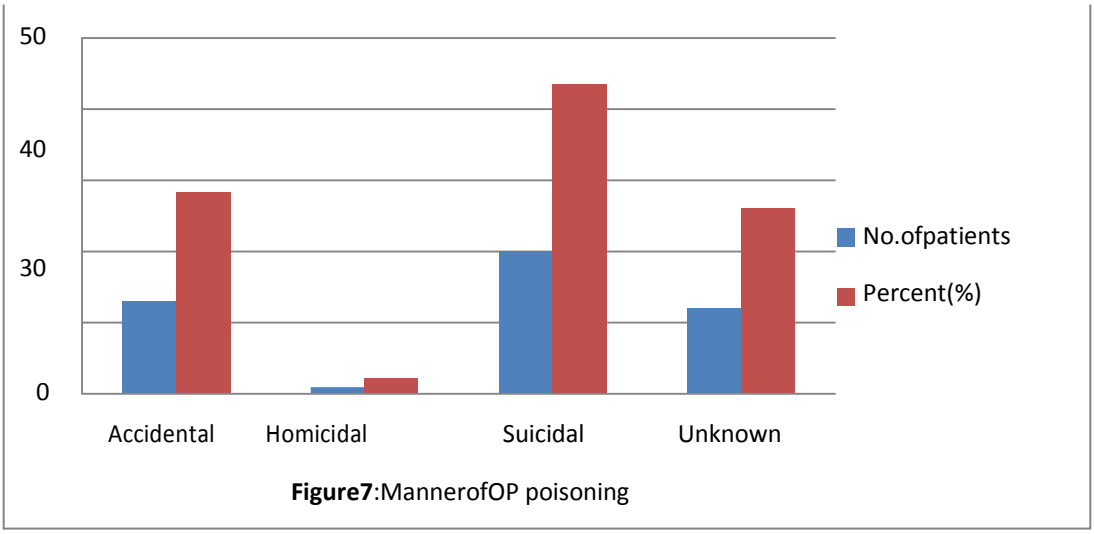


Figure7:MannerofOP poisoning

Table8andfigure8depictrouteofexposure,anddenotesthatoralroute(91.3%)wasmostcommonrouteofOPexposure.Inhalational(4.3%) andTopical(4.3%)beingrarelyusedroute ofexposure,whichwere mostlyduetoaccidentalmannerduringOP use.

Route of OP exposure	No. ofpatients	Percent (%)
Inhalational	2	4.3
Oral	42	91.3
Topical	2	4.3
Total	46	100.0

Table8:**Route of OPexposure**

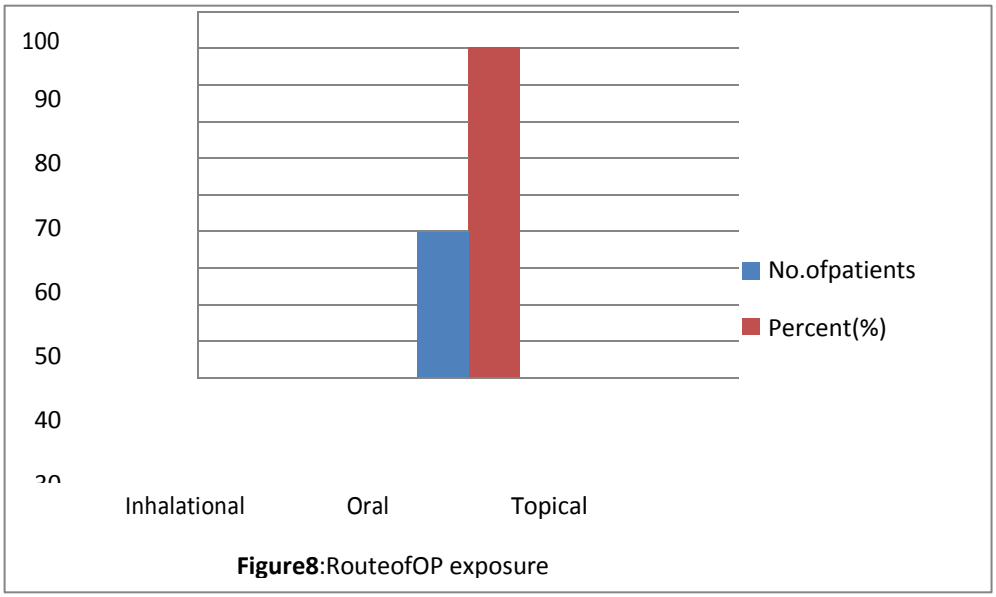


Figure8:RouteofOP exposure

Table9andfigure9describevariousreasonsofconsumingOPpoison,mostcommonbeingdue toalcoholinfluence(21.7%),followedbyfamilystress (13.0%).

Reason of consuming OP poison	No. ofpatients	Percent (%)
Consumedmistakenly as coughsyrup	1	2.2
Depression	1	2.2
Examphobia	1	2.2
Familystress	6	13.0
Financial stress	2	4.3
Homicidal	1	2.2
Loneliness	1	2.2
Mental illness	1	2.2
Relationshipproblem	1	2.2
Under influenceofalcohol	10	21.7
Unemployment	1	2.2
Work stress	1	2.2
Unspecified	19	41.3
Total	46	100.0

Table9:**Reason of consuming OP poison**

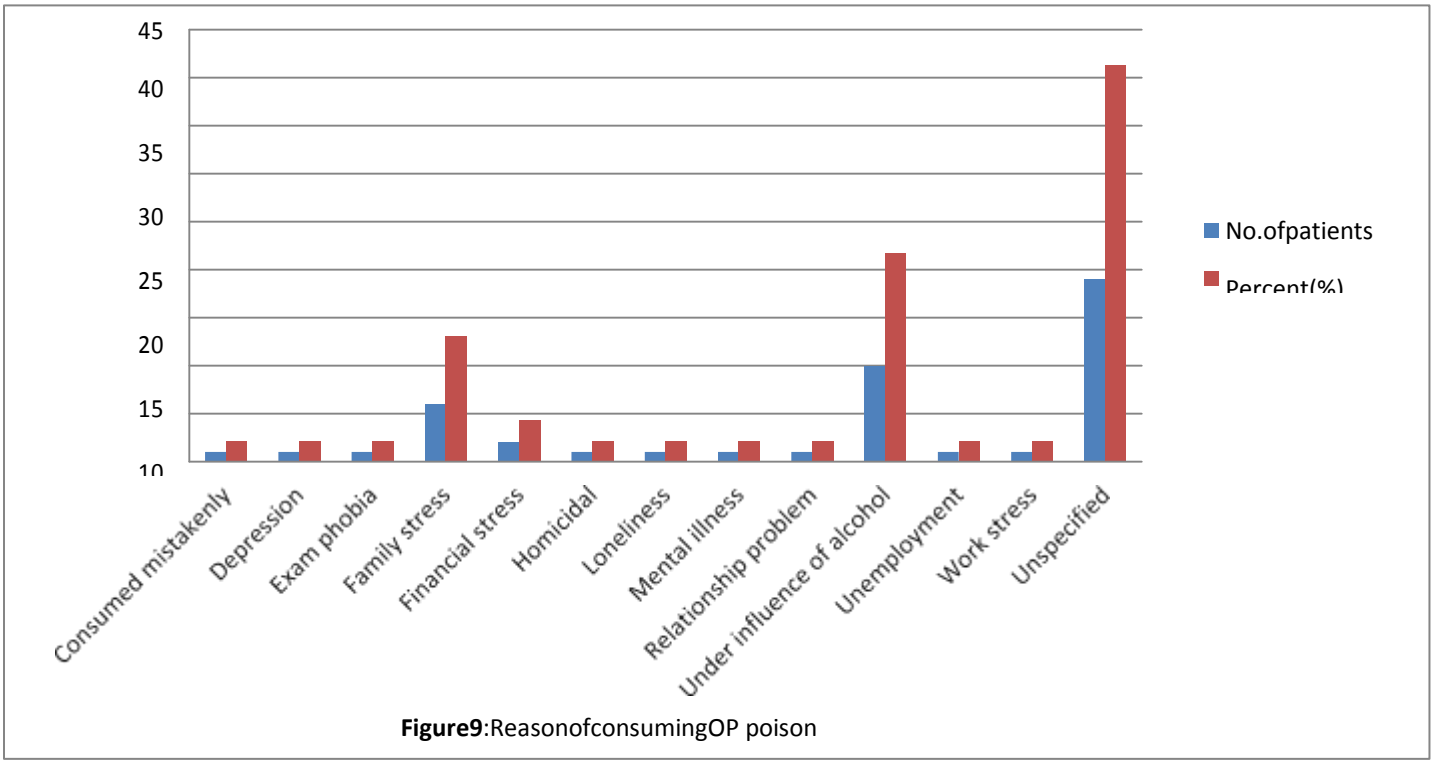


Figure9:ReasonofconsumingOP poison

Table10andfigure10describedistributionofOPpoisoningcasesinvariousrangesofSerumCholinesteraseLevel(U/L).Themean serumcholinesteraselevelis4491±4128U/L.Majorityofpatientshadserumcholinesteraselevel in therangeof 1000-2000 U/L (32.6%).

Serum CholinesteraseLevel(U/L)	No. ofpatients	Percent (%)
<1000	1	2.2
1000-2000	15	32.6
2001-3000	2	4.3
3001-4000	2	4.3
4001-5000	0	0.0
>5000	10	21.7
Unknown	16	34.8
Total	46	100.0

Table10:**Serum Cholinesteraselevel(U/L)**

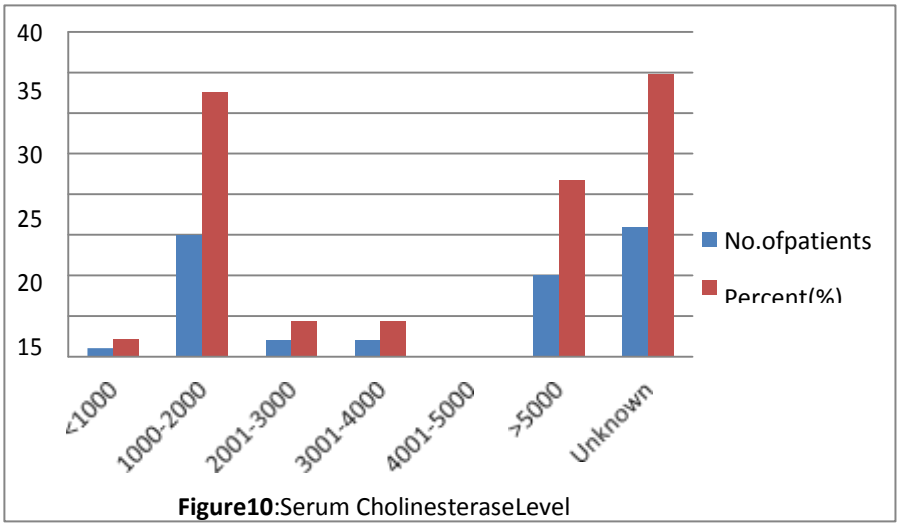


Figure10:Serum CholinesteraseLevel

Table11andfigure11describehospitalstayofOPpoisoningpatients.Themeanhospitalstaywas5.095±4.689days.Majorityofpatientswer ecompletelytreatedwithin5days(47.8%) and only2patients(4.3%)required morethan10daysfortreatment.

Hospital Stay(days)	No. ofpatients	Percent (%)
<5	22	47.8
5-10	18	39.1
>10	2	4.3
Unknown	4	8.7
Total	46	100.0

Table11:**Hospitalstayduration**

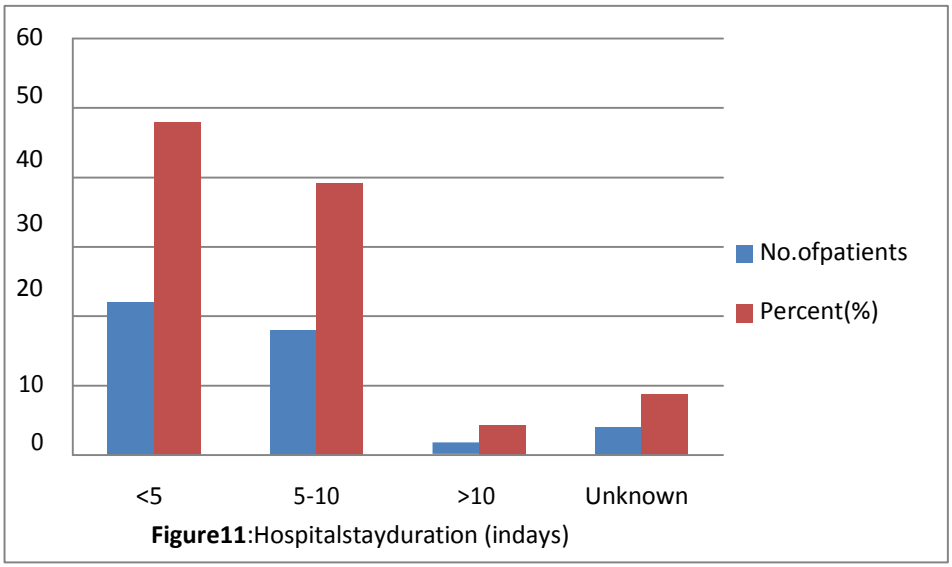


Figure11:Hospitalstayduration (indays)

Table12andfigure12describedurationofventilationgiventoOPpoisoningpatients.Themeandurationofventilationgivenwas67.4±62.2h ours.Only12patients(26.1%)weregivenventilatorsupport,amongwhich5patients(10.9%)neededventilationformorethan72hours as breathlessness andrespiratoryfailure is verycommon inOP poisoning.

Duration of Ventilationgiven (in hours)	No. ofpatients	Percent (%)
<24	4	8.7
24-72	3	6.5
>72	5	10.9
Not given	34	73.9
Total	46	100.0

Table12:**Duration ofventilationgiven**

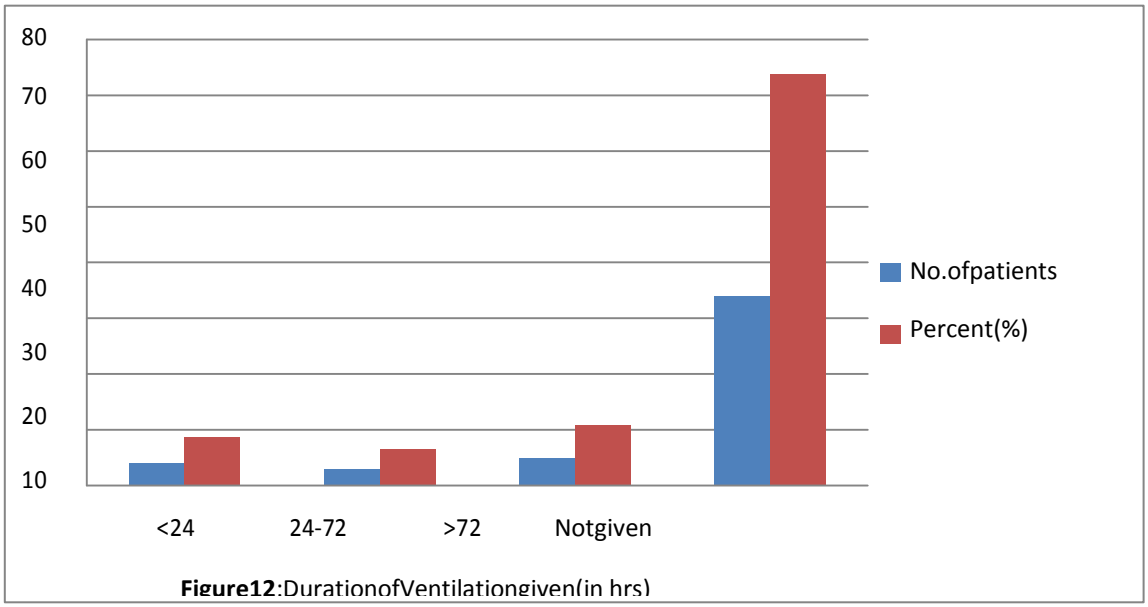


Figure12:DurationofVentilationgiven(in hrs)

Table13andfigure13describetimeelapsedbetweenOPpoisoningandFirstAidgiventothepatient.Themeantimeelapsedbetween OPpoisoningandFirstAidgivenwas2.782

±1.698hours.Majorityofvictimsgotfirstaidwithin2-4hours(30.4%)ofOPpoisoning.While6patients(13.0%)gotfirst aidaftermorethan4 hoursof OP poisoning.

TimebetweenOPpoisoning and FirstAidgiven (in hours)	**No. ofpatients**	**Percent (%)**
<2	10	21.7
2-4	14	30.4
>4	6	13.0
Unknown	16	34.8
Total	46	100.0

Table13:**Timebetween OPpoisoning andFirstAidgiven**

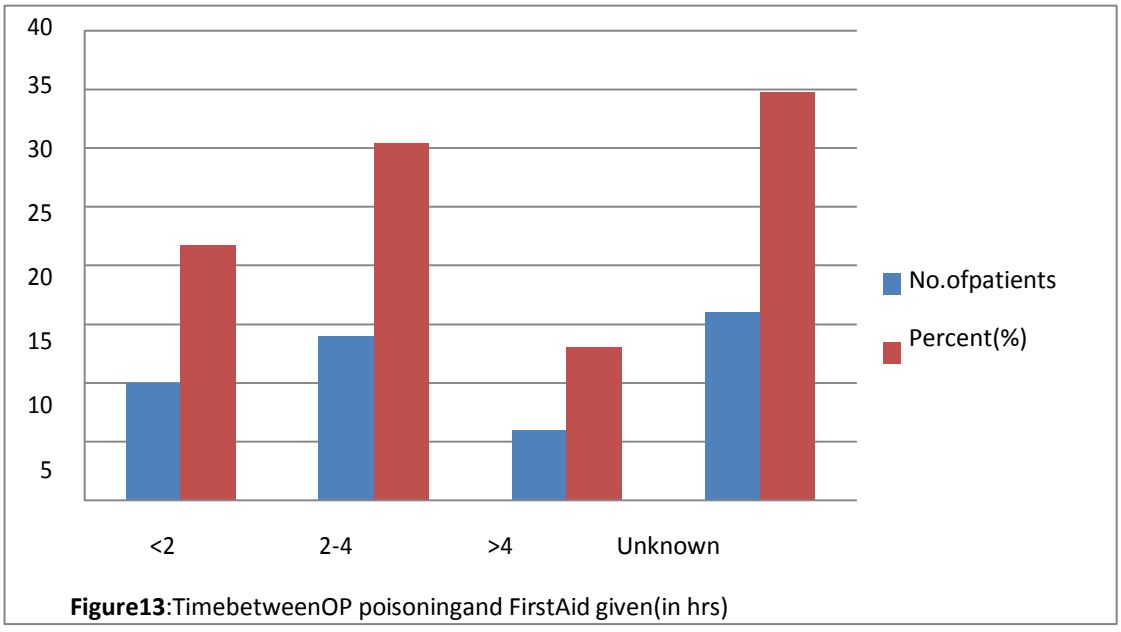


Figure13:TimebetweenOP poisoningand FirstAid given(in hrs)

Table14andfigure14describetimeelapsedbetweenOPpoisoningandHospitaladmissionofpatient.ThemeantimeelapsedbetweenOPpoisoningandHospitaladmissionwas3.801±1.679hours.Majorityofvictimsgotadmittedtothehospitalwithin2-4hours(43.5%) of OPpoisoning,while13 patients(28.3%) tookmore than4hours.

Timebetween OP poisoningand hospitaladmission (inhours)	**No. ofpatients**	**Percent (%)**
<2	5	10.9
2-4	20	43.5
>4	13	28.3
Unknown	8	17.4
Total	46	100.0

Table 14:**Timebetween OPpoisoning andhospital admission**

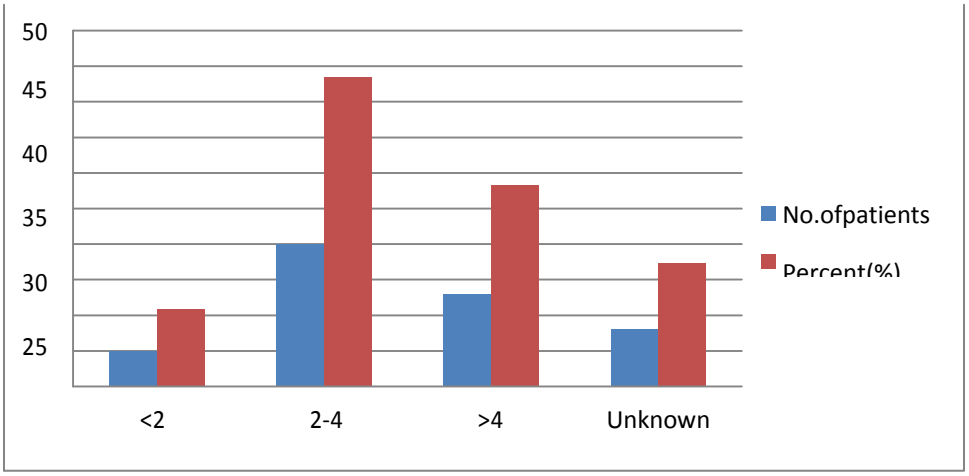


Table15andfigure15describetreatment'soutcome.Majorityofpatientsrecovered(71.7%)duetopropertreatmentandmanagementofOPpoisoning,while6Patients(13.0%)diedduringtreatmentand4patients(8.7%)tookDischargeAgainstMedicalAdvice(DAMA)and3patients(6.5%)werebroughtdeadtothehospital.

Treatment's outcome	**No. ofpatients**	**Percent (%)**
Recovered	33	71.7
Died	6	13.0
DAMA	4	8.7
Broughtdead	3	6.5
Total	46	100.0

Table15:**Treatment'soutcome**

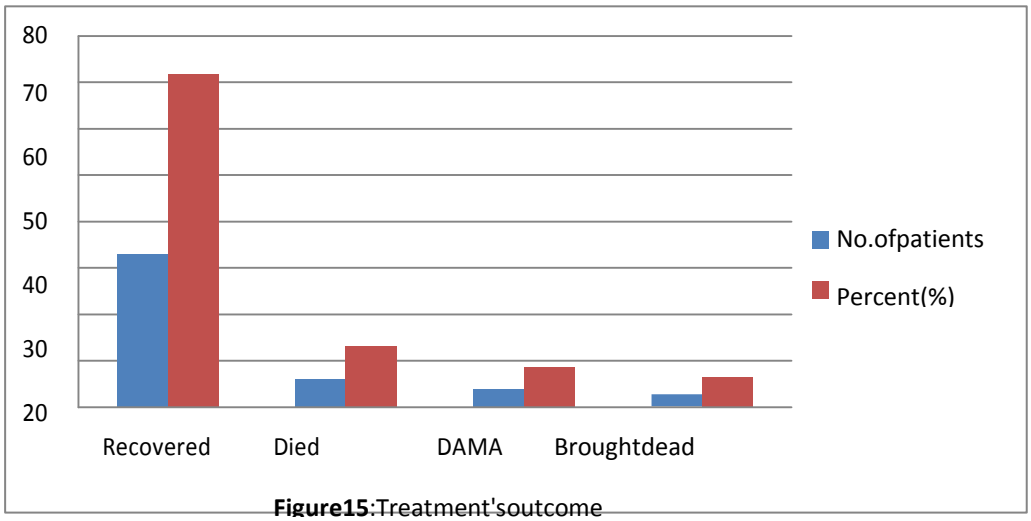


Figure15:Treatment'soutcome

Table 16andfigure 16describeno. ofpatientsgivenGastricLavage. 36 patients(78.3%)weregivengastriclavageinhospital,2patients(4.3%)werenotgivengastriclavageasthetimeelapsedfromconsuming OPwas too high, thatitwasofnouse.

GastricLavagegiven	**No. ofpatients**	**Percent (%)**
Yes	36	78.3
No	2	4.3
Unknown	8	17.4
Total	46	100.0

Table16:**GastricLavagegiven**

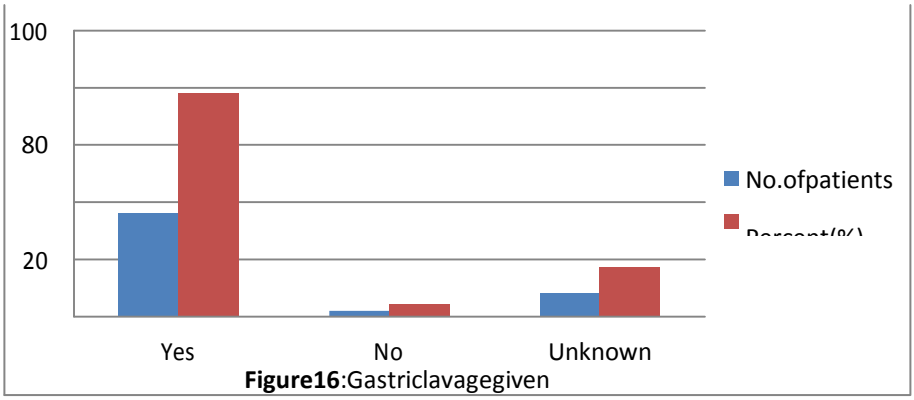


Figure16:Gastriclavagegiven

Table17andfigure17describeno.ofpatientsreferredtoPsychiatrydepartmentforcounseling.25patients(54.3%)werereferredtopsychiatr ybecausetheyattemptedsuicideduetopsychiatricreasonsandweretreatedaccordingly.8patients(17.4%)werenotreferredtoPsychiatryas theywere accidentalcasesandneedednopsychiatriccounseling.

Referred to Psychiatry	**No. ofpatients**	**Percent (%)**
Yes	25	54.3
No	8	17.4
Unknown	13	28.3
Total	46	100.0

Table17:**Referred toPsychiatry**

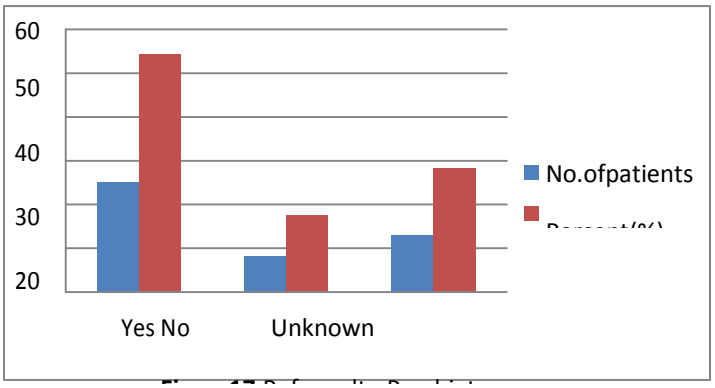


Table18andfigure18describeheartratevariationsamongpatientsofOPpoisoning.ThemeanHeart rateis89.41±15.89beats/min.32patients(69.6%)had normal rate,2 patients (4.3%)hadbradycardia.2patients(4.3%)hadefeebleheartrateand3patients(6.5%)hadheart ratewhichwas not palpable. Tachycardiawasseenin7patients(15.2%)becauseofatropineadministrationwasdoneduringfirstaid tothepatient.

Tachycardia=>100beats/minBradycardia =<60beats/minNormalrate = 60-100beats/min

Heart Rate	No. ofpatients	Percent (%)
Tachycardia	7	15.2
Bradycardia	2	4.3
Normal rate	32	69.6
Feeble	2	4.3
Not palpable	3	6.5
Total	46	100.0

Table18:**Heart Ratevariations**

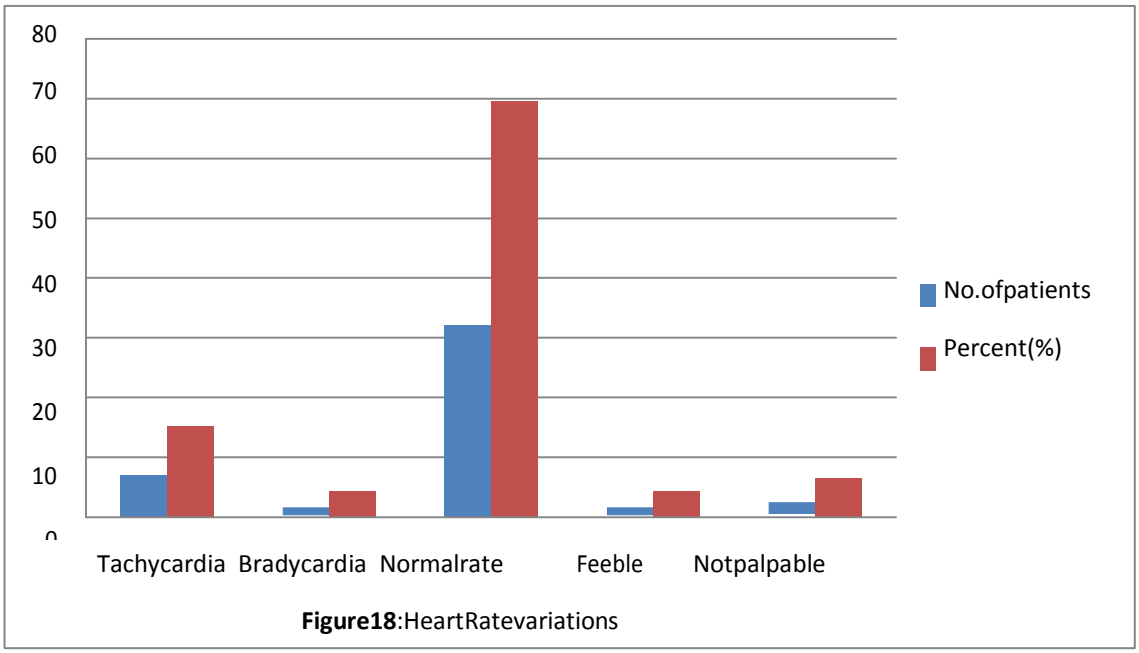


Figure18:HeartRatevariations

Table19andfigure19describeRespiratoryratevariationsamongpatientsofOPpoisoning.ThemeanRespiratoryrateis21.645±2.259breaths/min.18patients(39.1%)hadtachypnea,followedby13patients(28.3%)withnormalrateand12patients(26.1%)hadbradypnea,3patients(6.5%)werebroughtdeadandhencenorespiratorysoundswereheard.

Tachypnea= >20breaths/minBradypnea= <12breaths/minNormalrate = 12-20breaths/min

RespiratoryRate	No. ofpatients	Percent (%)
Tachypnea	18	39.1
Bradypnea	12	26.1
Normal rate	13	28.3
No respiratorysounds	3	6.5
Total	46	100.0

Table19:**RespiratoryRate variations**

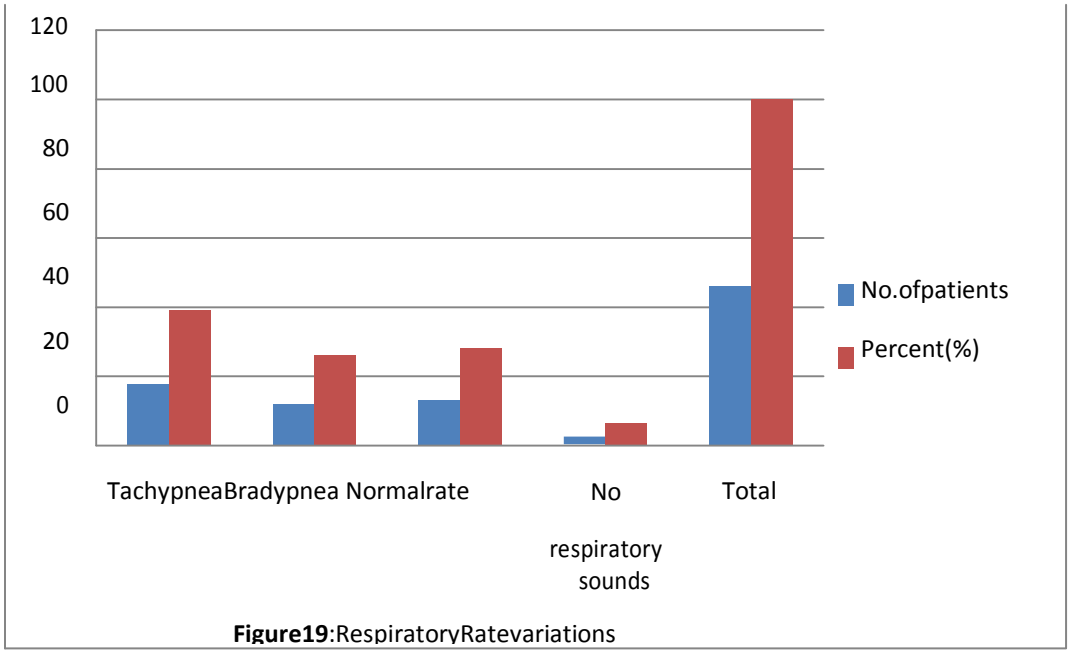


Figure19:RespiratoryRatevariations

Table20andfigure20describecommonclinicalfeaturesamongvictimsofOPpoisoning.65.1%victimssufferedfromvomitingandwasthemostcommonclinicalfeature,followed byGiddiness(46.5%) andConstrictedpupils(41.3%).

ClinicalFeature	Percent (%)
AlteredBehavior	14
Breathlessness	30.2
Unconsciousness	17.4
Nausea	20.9
Constricted pupils(miosis)	41.3
Giddiness	46.5
Vomiting	65.1

Table20:CommonClinicalfeaturesamongstvictims of OPpoisoning

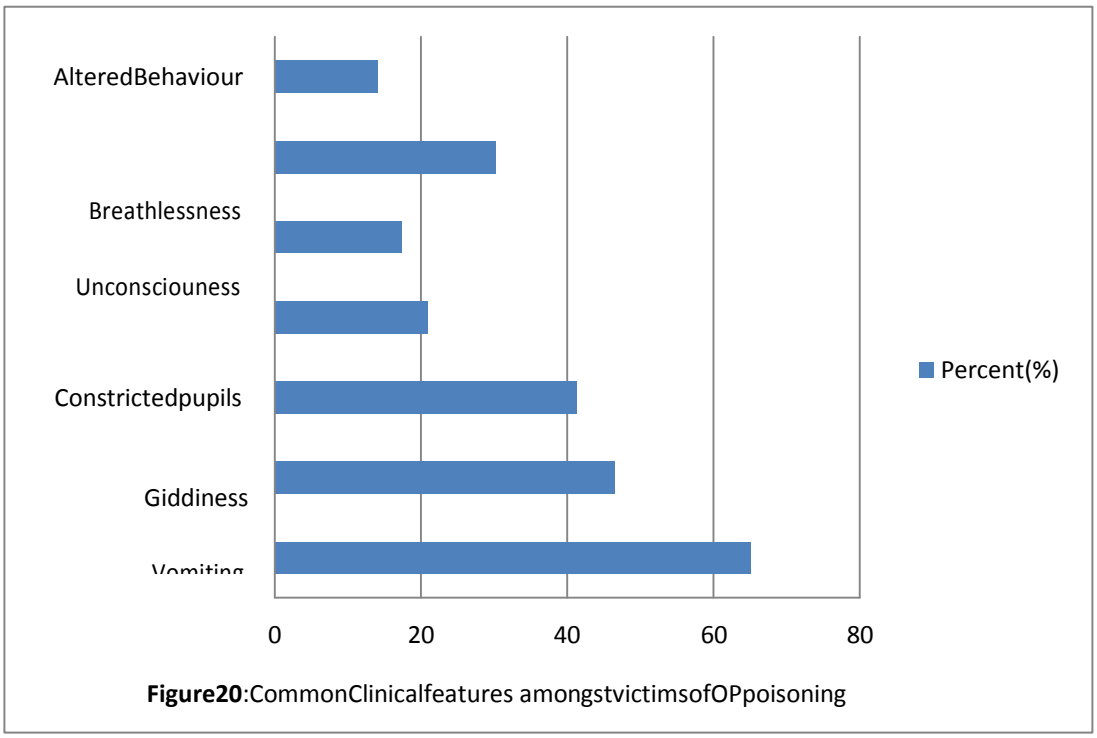


Figure20:CommonClinicalfeatures amongstvictimsofOPpoisoning

Table21andfigure21describecorrelationbetweenserumcholinesteraselevelandhospitalstayduration.Thep-valueis**0.074**,hencethisshows**highlysuggestive**correlationbetweenthesetwoparameters.

(U/L)	<5	5-10	>10	Unknown	
<1000	0	1	0	0	1
1000-2000	5	9	1	0	15
2001-3000	2	0	0	0	2
3001-4000	0	2	0	0	2
4001-5000	0	0	0	0	0
>5000	9	1	0	0	10
Unknown	6	5	1	4	16
Total	22	18	2	4	46

Table21:**CorrelationbetweenSerumcholinesteraselevel and Hospitalstayduration**

10

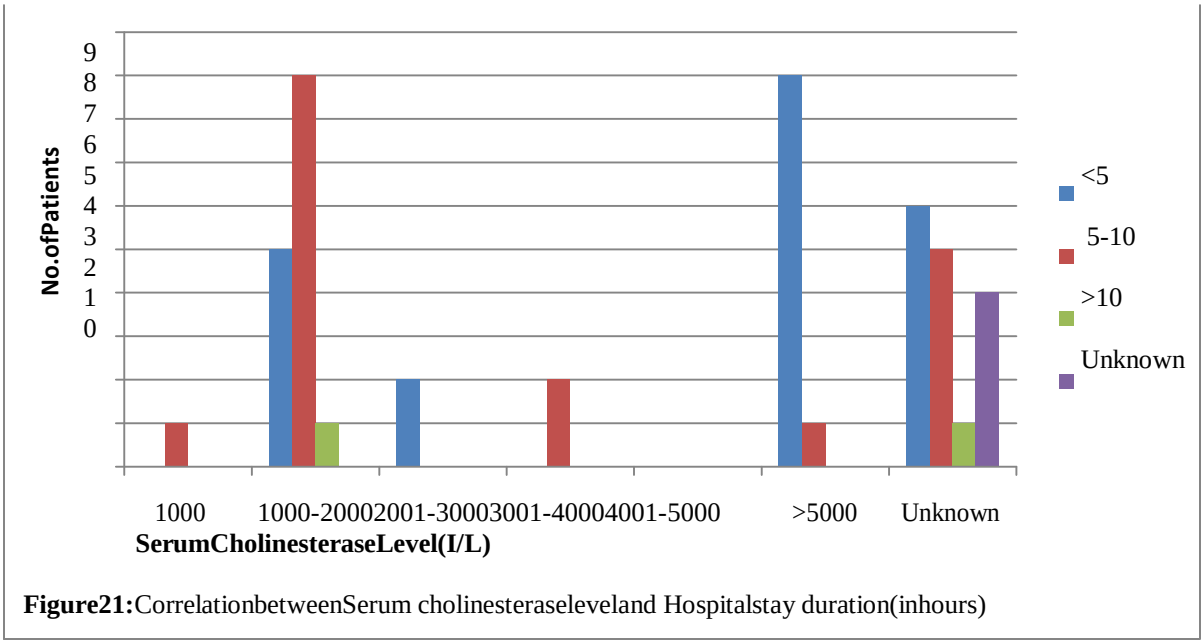


Figure21: CorrelationbetweenSerum cholinesteraseleveland Hospitalstay duration(inhours)

Table22andfigure22describecorrelationbetweenserumcholinesteraselevelandduration ofventilationgiventopatients.Thep-valueis**0.04**,hencethisshows**Significant**correlationbetweenthesetwoparameters.

A. Serum Cholinesterase

	Duration of Ventilationgiven (in hours)				Total
Level(U/L)	**<24 hr**	**24-72 hr**	**>72 hr**	**Notgiven**	
<1000	0	1	0	0	1
1000-2000	2	1	4	8	15
2001-3000	1	0	0	1	2
3001-4000	1	0	0	1	2
4001-5000	0	0	0	0	0
>5000	0	0	0	10	10
Unknown	0	1	1	14	16
Total	4	3	5	34	46

Chi-square = 33.497, p =0.04 (Significant)

B. Table 22: CorrelationbetweenSerumcholinesteraselevel and duration ofVentilationgiven

No.ofPatients

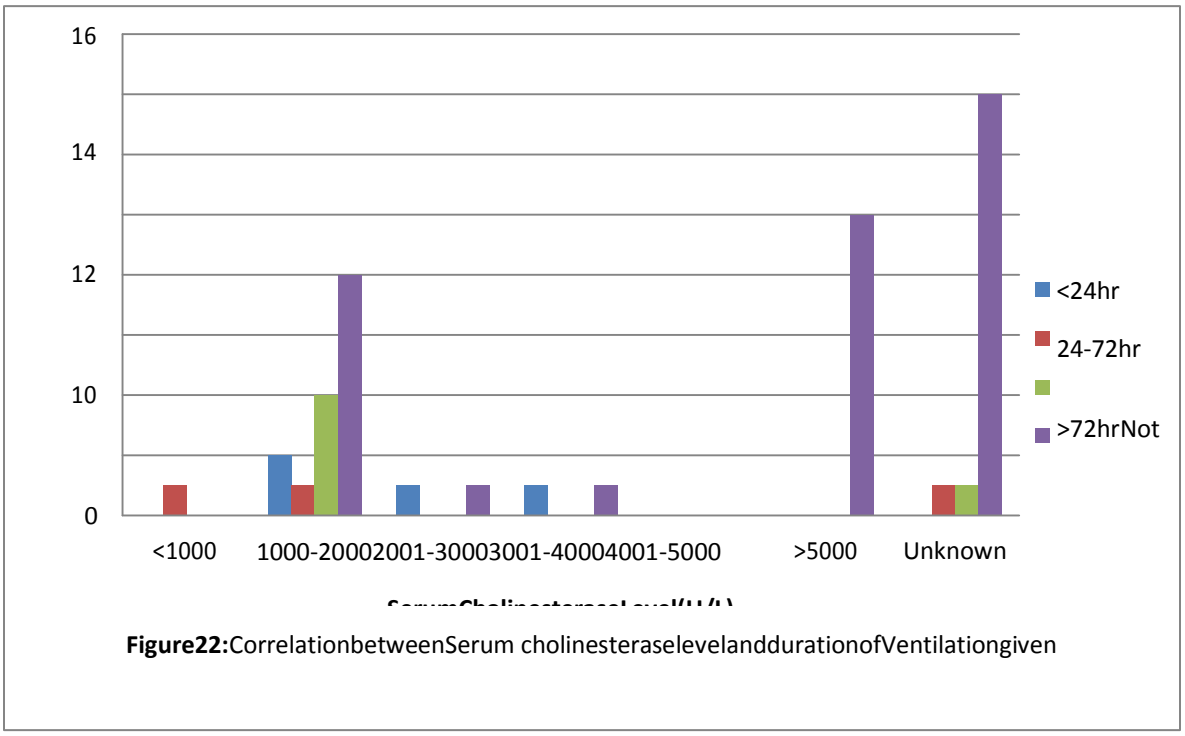


Figure22: CorrelationbetweenSerum cholinesteraselevelanddurationofVentilationgiven

Table23andfigure23describecorrelationbetweenserumcholinesteraselevelandtreatment'soutcome.Thep-valueis**0.136**,hencethisshows**nosignificant**correlationbetweenthesetwoparameters.

C. Serum

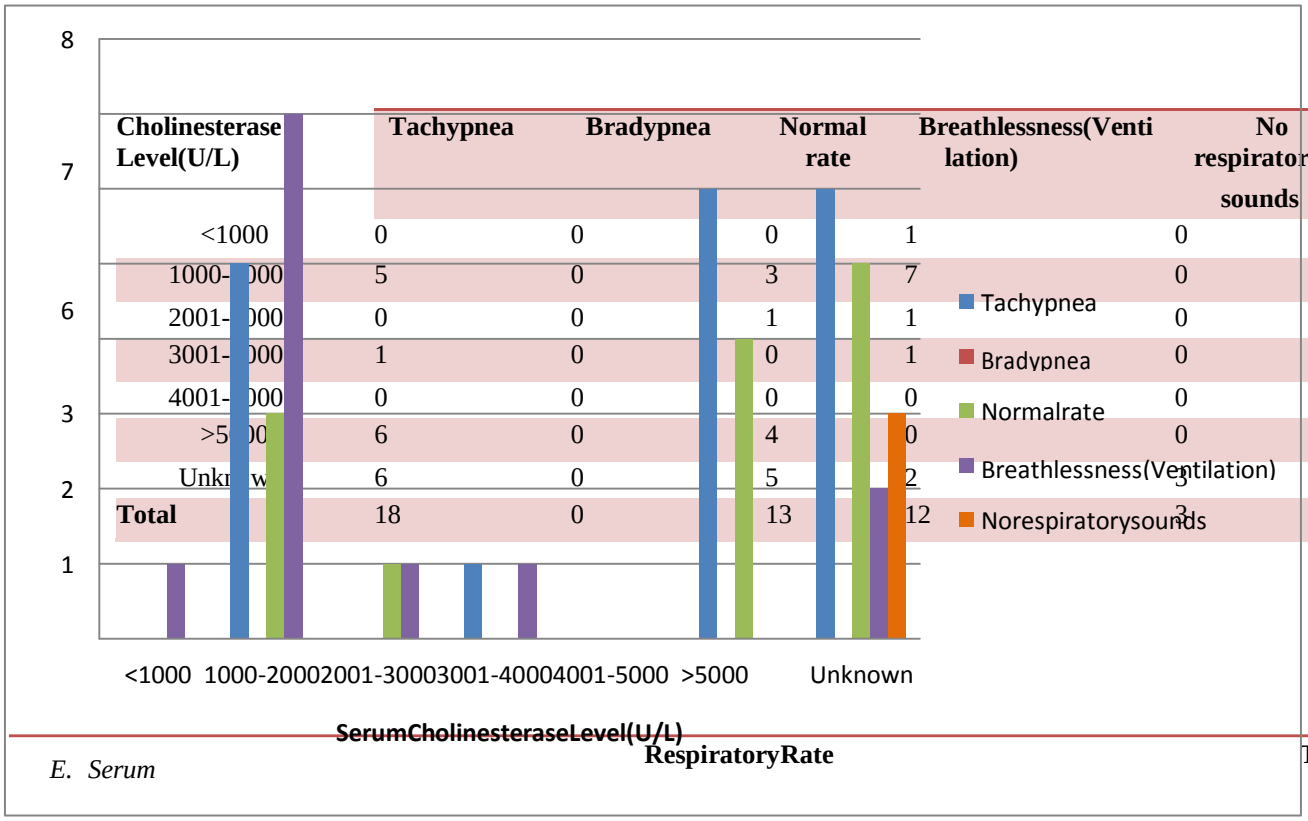
Cholinesterase Level(U/L)	Treatment's outcome				Total
	Recovered	Died	DAMA	Brought dead	
<1000	1	0	0	0	1
1000-2000	10	5	0	0	15
2001-3000	1	1	0	0	2
3001-4000	2	0	0	0	2
4001-5000	0	0	0	0	0
>5000	9	0	1	0	10
Unknown	10	0	3	3	16
Total	33	6	4	3	46

No.ofPatients

D. Chi-square = 21.030, p =0.136(Not Significant)

Table 23: CorrelationbetweenSerumcholinesteraselevel and Treatment'sOutcome

Table24andfigure24describecorrelationbetweenserumcholinesteraselevelandRespiratoryratevariations.Thep-valueis**0.199**,hencethisshows**nosignificant**correlationbetweenthesetwoparameters.



Cholinesterase Level(U/L)	Tachypnea	Bradypnea	Normal rate	Breathlessness(Ventilation)	No respiratory sounds	
<1000	0	0	0	1	0	1
1000-2000	5	0	3	7	0	15
2001-3000	0	0	1	1	0	2
3001-4000	1	0	0	1	0	2
4001-5000	0	0	0	0	0	0
>5000	6	0	4	0	0	10
Unknown	6	0	5	2	3	16
Total	18	0	13	12	3	46

E. Serum

F. Chi-square = 19.342, p =0.199(Not Significant)

Table 24:CorrelationbetweenSerumcholinesteraselevel and RespiratoryRatevariations

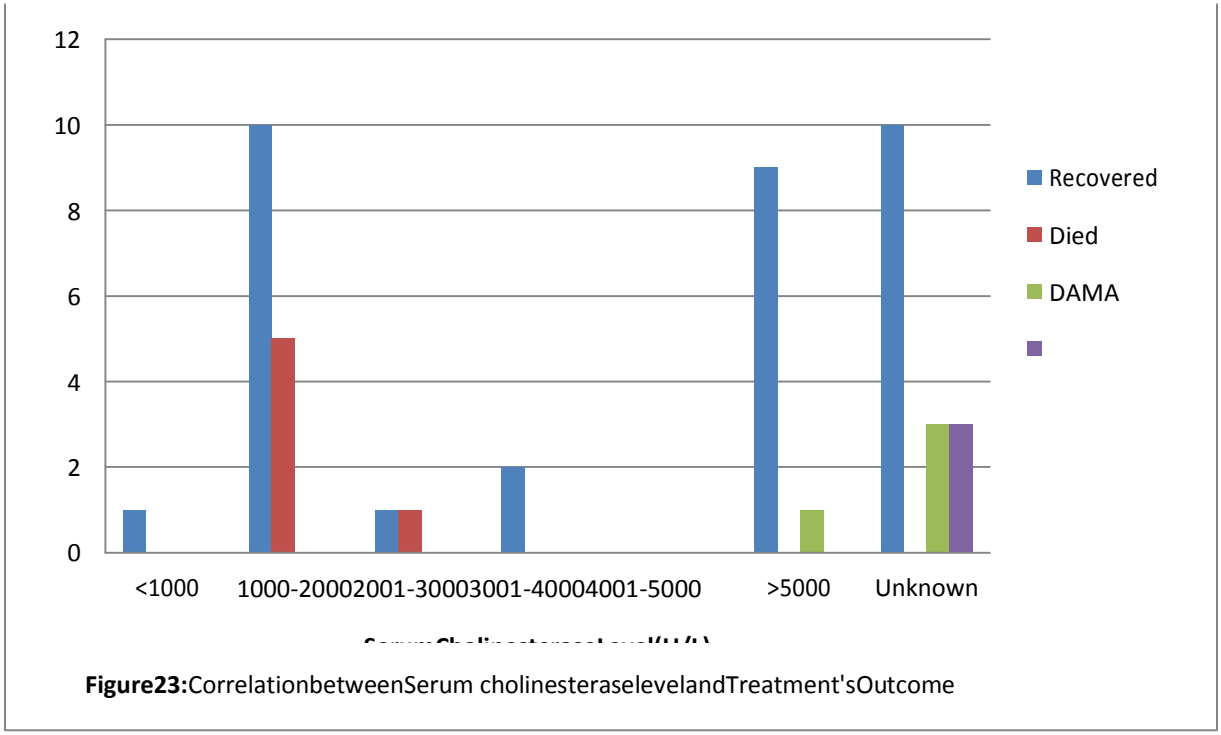


Figure23: CorrelationbetweenSerum cholinesteraselevelandTreatment'sOutcome

Table25andfigure25describecorrelationbetweenserumcholinesteraselevelandbreathlessnessinpatients.Thep-valueis**0.142**,hencethisshows**nosignificant**correlationbetweenthesetwoparameters.

Serum CholinesteraseLevel(U/L)	Breathlessness Yes	No	Total
<1000	1	0	1
1000-2000	7	8	15
2001-3000	1	1	2
3001-4000	1	1	2
4001-5000	0	0	0
>5000	1	9	10
Unknown	2	11	13
Total	13	30	43

G. Chi-square = 8.269,p=0.142(Not Significant)

H. Table 25:CorrelationbetweenSerumcholinesteraselevel andBreathlessnessamong OP poisoning patients

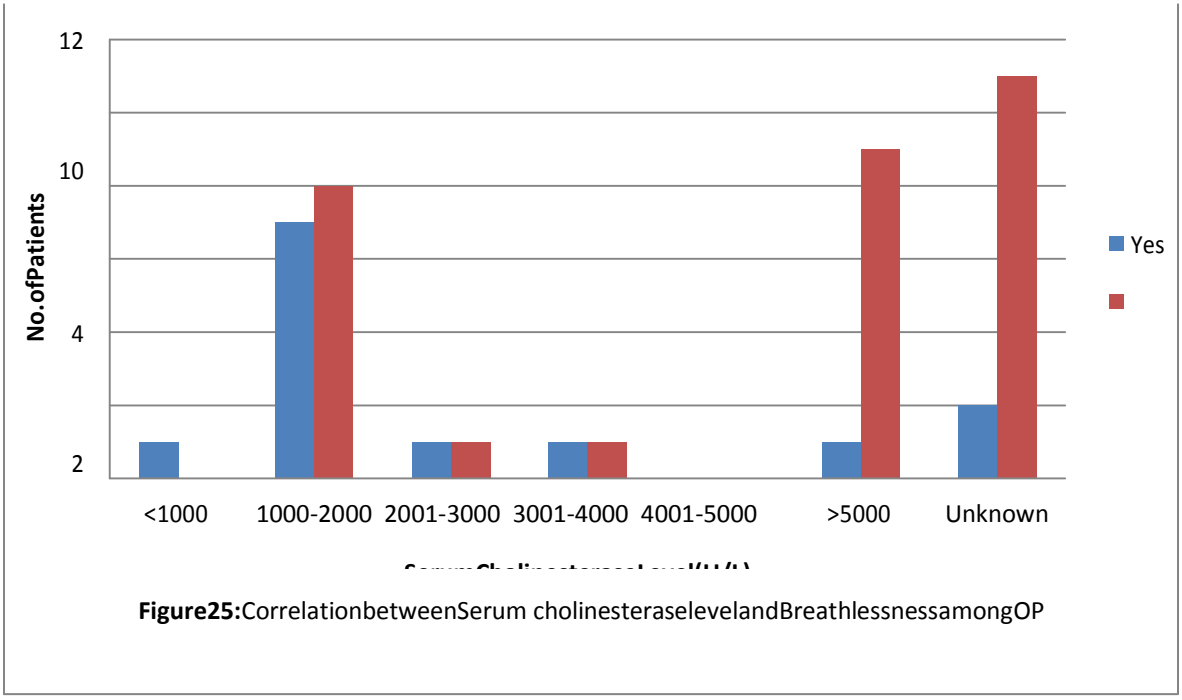


Figure25: CorrelationbetweenSerum cholinesteraselevelandBreathlessnessamongOP

Table26andfigure26describecorrelationbetweenserumcholinesteraselevelandvomitingamongpatients.Thep-valueis**0.338**,hencethisshows**nosignificant**correlationbetweenthesetwoparameters.

Serum CholinesteraseLevel(U/L)	VOMITING Yes	No	Total
<1000	0	1	1
1000-2000	12	3	15
2001-3000	2	0	2
3001-4000	1	1	2
4001-5000	0	0	0
>5000	5	5	10
Unknown	8	5	13
Total	28	15	43

I. Chi-square = 5.681,p= 0.338(Not Significant)

J. Table26:CorrelationbetweenSerumcholinesteraselevel and Vomitingamong OP poisoning patients

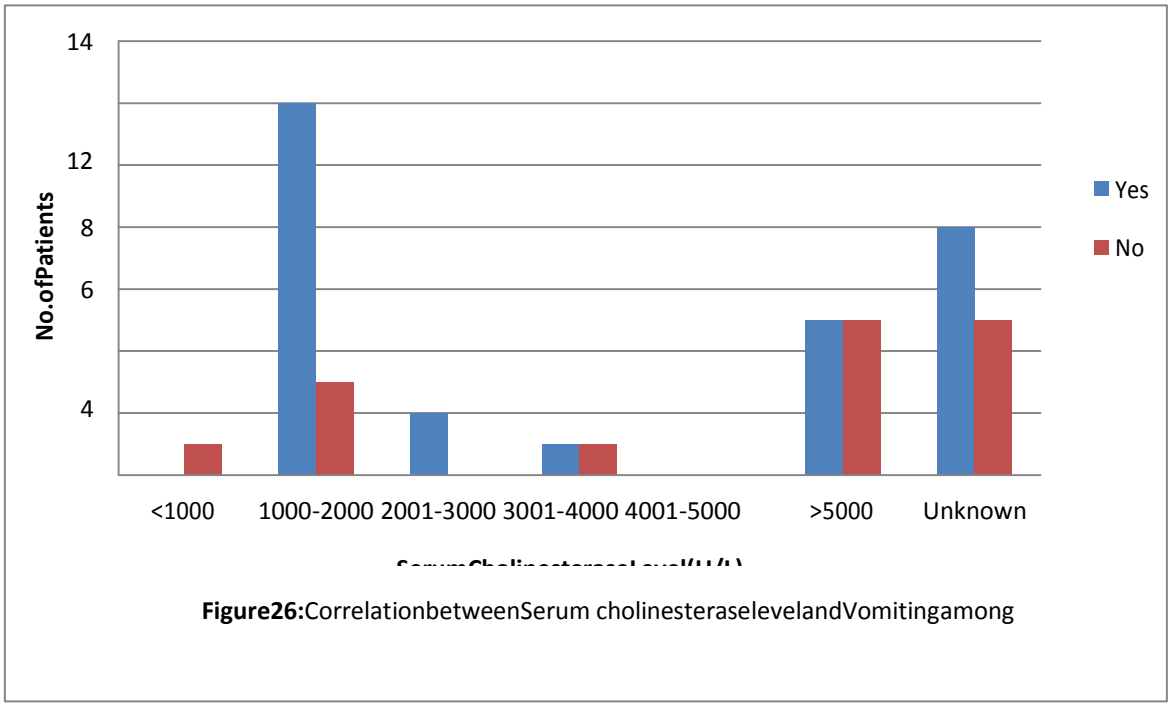


Figure26: CorrelationbetweenSerum cholinesteraselevelandVomitingamong

Table27andfigure27describecorrelationbetweenserumcholinesteraselevelandunconsciousnesspatients.Thep-valueis**0.799**,hencethisshows**nosignificant**correlationbetweenthesetwoparameters.

Serum CholinesteraseLevel(U/L)	Consciousness No	Yes	Total
<1000	0	1	1
1000-2000	4	11	15
2001-3000	0	2	2
3001-4000	0	2	2
4001-5000	0	0	0
>5000	1	9	10
Unknown	3	13	16
Total	8	38	46

K. Chi-square = 2.352,p= 0.799(Not Significant)

L. Table 27:CorrelationbetweenSerumcholinesteraselevel andConsciousnessamong OP poisoning patients

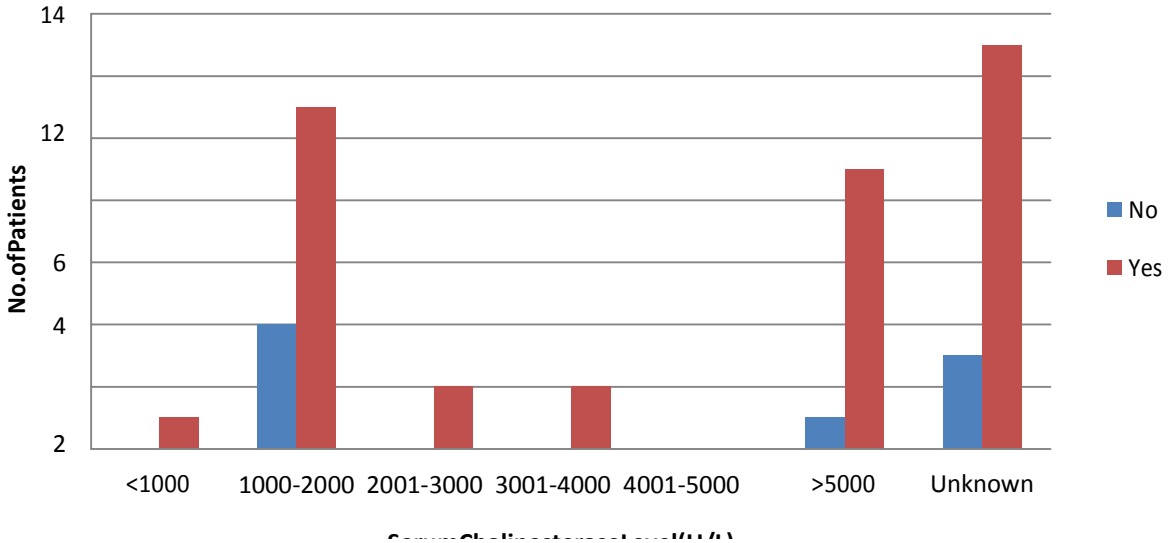


Figure27: CorrelationbetweenSerum cholinesteraselevelandConsciousnessamongOP

Table28andfigure28describecorrelationbetweentimeelapsedbetweenOPpoisoningandhospitaladmissionversustreatment's outcome.Thep-valueis**0.062**,hencethisshows**highlysuggestive**correlation betweenthesetwo parameters.

M. Timebetween OPpoisoning and hospitaladmission (in hours)	Treatment's outcome				Total
	Recovered	Died	DAMA	Broughtdead	
<2	5	0	0	0	5
2-4	16	1	2	1	20
>4	10	3	0	0	13
Unknown	2	2	2	2	8
Total	33	6	4	3	46

N. Chi-square = 16.239, p =0.062(HighlySuggestive)

Table28:CorrelationbetweenTimeelapsedbetweenOPpoisoning and hospitaladmissionversus Treatment's outcome

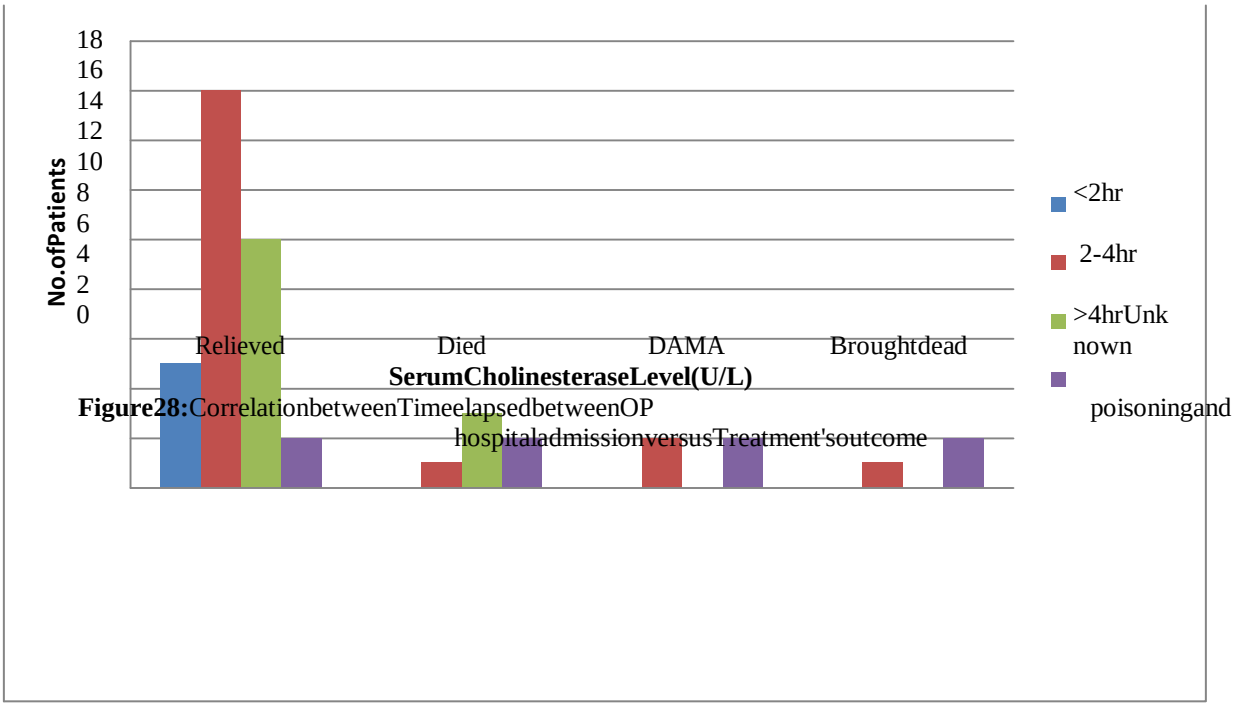


Figure28: CorrelationbetweenTimeelapsedbetweenOP poisoningand hospitaladmissionversusTreatment'soutcome

Table29DescribesDescriptivestatistics.

Variable	No. of patients	Mean	Median	Standard Deviation	Minimum	Maximum
Age(inyears)	46	29.71	25	12.917	0.67	69
Serum CholinesteraseLevel(U/L)	30	4491	1917	4128	989	14186
Hospital stayduration (Days)	42	5.095	4	4.689	1	29
Duration ofventilationgiven(in hours)	12	67.4	47.8	62.2	1.8	149.5
Timeelapsedbetween OPpoisoning and firstaidgiven(in hours)	30	2.782	2.685	1.698	0.4	8
Timeelapsedbetween OPpoisoning and hospitaladmission(inhours)	38	3.801	3.535	1.679	1.05	8.85
RespiratoryRate (breaths/min)	31	21.645	22	2.259	16	28
Heart rate(beats/min)	41	89.41	90	15.89	42	130

Table30:**Descriptive Statistics**

II. DISCUSSION:

Thepresentstudyisbasedondatacollectedfrom July 1st 2017 to December 31st 2017. A totalof 46 caseswere studiedwho were admittedfor treatmentof OP poisoninginrespectivetertiarycarehospital.Theclinicalanddiagnosticfindingsofthisstudyarecomparedwithstudiesinreviewofliterature.

Patternofpoisoninginaregiondependsonvarietyoffactorssuchaseasyaccesstothe poisons,SEstatusofthepopulation andreligious/culturalinfluences.

A. Epidemiologicalfactors / Socio-demographicfactors:

4. **Seasonalvariation:**Inourstudy,incidenceofOPpoisoningwashighestduringOctober.Foraccurateseasonalvariation,astudyshouldbeconductedwhichwouldincludeminimum1wholeyear,unlikepresentstudyincludingonly6months.
5. **Ageof patients:**Inourstudy, majorityofpatients were in the age group of16-30years(67.4%).Asthisagegroupisthemainworkingpopulation,sotheyaremorepronetostressandaccidentalexposure.Thisisinagreementtostudiesdonebymishraetal[16]andKumaretal[17].Themeanageinthisstudyis29.71 ±12.917years.The ageofpatientsvaried from8 monthsto69years.
6. **Genderdistribution:**Thisstudyrevealedthattherewasmalepredominance78.3%,andfemaleaccountingforthere maining21.7%.Themaletofemaleratiointhisstudyis3.6:1.Thisisbecausemalesarethemajorworkinggroupofsocietyin India.This corresponds to genderdistributionreported byPatelet al[18],Shettietal[19]andJoshi etal[1].
7. **Maritalstatus:**34.8%patients inour studywere unmarried.
8. **Area:**Majorityofcaseswerefromruralarea(91.3%),urbancaseswereonly8.7%.Thisfindingcorrespondswithstudiesof Mishraetal[16],Pateletal[18]andJoshietal[1].UseoftheOPcompoundsismoreinruralareasthaninurbanbecauseoftheirutilityasinsecticides,pesticidesandfungicidestoprotecttheagriculturalcrops.
9. **TypeofOPpoison:**VarietyofOPcompoundwereseenusedduringthestudy,vizDicholorvos,Dimethoate,Chlorpyrifos,Glyphosatebeingmostcommonamongall.
10. **Mannerofpoisoning:**Thisstudyrevealedthatmajorityofcases ofOPpoisoningweresuicidalcases(43.5%),followedbyaccidental(28.3%)andhomicidalbeingleast(2.2%).Thisisinagreementtostudiesdonebymishraetal[16],Patel etal[18]andJoshietal[1].Accidentalpoisoninginourstudywasmainlyseenduetoconsumptionof OP under alcohol influence.

11. **RouteofOPexposure:** AlmostallpatientsusedoralrouteasOPexposure91.3%.Topicalandinhalationalroutesaccountedforonly4.3%respectively,which are accidental cases.
12. **Reasonofconsuming:** Variousreasonscontributedtotheconsumptionof OPpoison.Majorityofsuicidalpoisoning wasduetofamilystress(13%),andmajority ofaccidentalpoisoning casescanbeattributedtoalcoholconsumption.
13. **Serumcholinesteraselevel:** AlterationinlevelsofSCEwerenotedinmajorityofcases.Mostcaseswerefoundtobeintherangeof1000-2000U/L(32.6%).The meanSCElevelis4491±4128U/L.
14. **Hospitalstay:** 47.8%patientsgotrecoveredinlessthan5daysofadmissionand39.1% patientsrequired 5-10daystorecover.
    Only2patients(4.3%)neededmorethan10daysfortreatment.Highesthospitalstaydurationbeing29days.The meanhospitalstaydurationbeing5.095±4.689days.
15. **Durationofventilationgiven:** Majorityofpatients(73.9%)werenotassistedwithventilation.Only5patientsreceivedventilationformorethan72hours.Highestdurationofventilationgivenwas149.5hours.Themeandurationofventilationwasgiven for67.4±62.2hours.
16. **TimeelapsedbetweenOPpoisoningandfirstaidgiven:** Mostofthepatientsreceivedfirstaidwithin2-4hoursofpoisoning(30.4%).10patients(21.7%)receivedfirstaidwithin2hourswhereas6patients(13%)gotfirstaid aftermorethan4hours.Themeantimeelapsedbetween OPpoisoningandfirstaidgivenwas2.782±1.698hours.
17. **TimeelapsedbetweenOPpoisoningandhospitaladmission:** Majoritypatientsgotadmittedtohospitalwithin2-4hoursofpoisoning(43.5%).13patients(28.3%)gotadmittedtothehospitalaftermorethan4hourswhereas5patients (10.9%)gotadmittedtohospitalwithinlessthan2hours.Themeantimeelapsedbetween OPpoisoning andhospitaladmission was3.801±1.679 hours.
18. **Outcomeoftreatment:** Majorityofpatientswererecoveredaccountingto71.7%.Only6patients(13%)diedduringthetreatment.4patientstookDischargeagainstMedicalAdvice(DAMA)and3patients(8.7%)werebroughtdead tothehospital.This is inagreementtostudiesdone byJoshiet al[1].
19. **Gastriclavage:** Almostallpatientsreceivedgastriclavage(78.3%).Gastriclavage leads to removal ofOP poisoningested bythe patients,whichwouldhavedeterioratedtheclinicalcourse.Onlytopicallyexposed,inhalationalandpatientswithhugetimedifferencebetweenOPconsumptionwerenotgivengastriclavage (17.4%).
20. **Psychiatriccounseling:** 54.3%patientsin ourstudywerereferredtopsychiatricdepartmentforcounseling.Allthepatientshavingorsuspiciousofsuicidalintentionweregivenpsychiatriccounseling.Alsosomepatientsofsuicidalintentionmayclaimittobeaccidental,hencepsychiatriccounselingisalsogiven

tosuchpatients.Onlypatientswithaccidentalandhomicidalmannerwerenotcounseled.

B. Clinicalfeatures:

21. **Heartratevariation:** Abigproportionofpatientshadanormalheartrateaccountingforabout69.6%.7patients(15.2%)experiencedtachycardiabecauseofatropineadministrationduringfirstaidtothepatient,4.3%ofthepatientsunderwentBradycardia,whereasonly4.3%hadfeebleheartrate.Therewerealso3patientsthatwerebroughtdeadhavinganon-palpableheartrate,itconstitutesfor4.3%. The meanheartratewas89.41±15.89beats/min.
22. **Respiratoryrate:** Outof thetotalpatientsabout13(28.3%)hadanormalrespiratoryrate.Tachypnoeawasexperiencedby18patients(39.1%).Asignificantno.ofabout12patients(26.1%)hadbradypnoeaandrequiredventilation.The3patientsbroughtdeadshowednosignsofrespiration.Themeanrespiratoryrate calculatedwas21.645±2.259breaths/min.
23. **CommonClinicalFeatures:** AmongstalltheclinicalfeaturesconcernedwithOPpoisoning,vomiting(65.1%)was themostcommon,thiscorrespondstostudiesbyMishraetal[16]andPateletal[18].Followedbygiddiness(46.5%)andconstrictedpupils(41.3%).Otherclinicalfeaturesthatthevictimswereenforced withincludednausea(20.9%),alteredbehavior(14%),breathlessness(30.2%) andunconsciousness(17.4%).

Conclusion:-

Indiapredominantlybeingagrariancountry,mostpeoplearebelowpovertylineandareengagedinagricultural work.Farmershavethehighestchanceofbeingatriskof OPpoisoningduringtheiroccupationalpractices.HenceOrganophosphoruspoisoningisoneofthemostcommonpoisoninginruralareas,predominantlyinyoungworkingpopulation(16-30years)withamalepredominanceandmostlyunmarried.Henceawarenessshouldberaisedamongsttheagricultural workersandyouthabouttheharmfulanddeleteriouseffectsofOPcompoundsandproperknowledgeshouldbeprovided.MonthofOctoberbeingthemostcommonseasonbringsthehigheststrainofOP poisoning casestothehospital.

DicholorvosandDimethoateweremostcommonlyconsumedOPpoison.Themainreasonforselectionof OPpoisonisthatitischeap,easilyavailableoverthecounterandusedasamajorpesticideduringfarming,soreadilyavailableathomesofthefarmers.Majorityofcasesconsumed OPpoisonwithsuicidalintentionsandbyoralroute.Thisisbecausewiththeincreasingstressinlife,suicideamongadolescentsandyoungadultsisacommonproblem.Familystressincaseofsuicidalpoisoningandalcoholinfluenceforaccidentalpoisoningemergedtobethemostcommoncauses.So,properguidelinesandprecautionsarenecessarytohandlethesepesticides.PatientwithintentionalOPpoisoningmustundergopsychiatricconsultationduringtheirstayinthehospital.Mortalityrateinthisstudywasonly13%,duetotheclosepro ximityofmedicalfacilities available inour region.

Themostcommonlyfoundclinicalfeaturewasvomiting.Giddinessandmiosiswerealsoseeninmanypatients.Mostofthepatientshadserumcholinesteraselevelintherangeof1000-2000U/L(32.6%).Mortalitywaslessamongthepatientswhowerepresentedtothehospitalearlyascomparedtothosewhopresentedlate.Upgradationoftheprimaryhealthcenterfacilitiestoprovideimmediatefirstaidfor OPpoisoning,couldconsiderablyinfluencetoreducebothmortalityandmorbidityduetoOPpoisoning.

Outcomeoftreatmentworsenedwithincreaseindaysofhospitalstay.Hospitalstaydurationwasmoreamongthepatientswhohadlowlevel(<2000U/L)ofserumcholinesteraseenzyme.Needforventilatorsupportwasseenmoreinpatientswithlow SCElevels(<2000U/L),whichrelateswiththestudydonebyGoswamyetal[20].Bychance,prediction oftreatment'soutcome,bloodpressure,respiratoryrate,breathlessness,vomitingandconsciousnesscannotbemadebasedonthelevelsofserumcholinesterase.Thissuggeststhecholinesteraseenzymelevelsdoesnotpredictthemortality rate.Similarresultsareobservedinastudyconductedby Hiremathet al[21]andShetti etal[19].

Toconclude,referenceofserumcholinesteraselevelestimationcanbeusedtogetanideaabouthospitalstayandddurationofventilationneededbythepatient.Apredictioncanbemadeabouttreatment'soutcomewiththehelpoftimeelapsedbetweenOPpoisoning&hospitaladmission.SCElevelcannotbeusedinevaluationofclinical courseofpatientandtreatment'soutcome.

Henceserumcholinesteraselevelshavenoprognosticvalueinacute OPpoisoning.Thus,SCElevelasanindicatortoidentifyhigh-riskpatientsbasedonthe measurementofSCEisalthoughusefulbutisnothighlyreliableduetopatientBMR/metabolismvariations.

ConflictofInterest: Nil

Ethics: There is on ethical violation in the conduction of study.

Funding: No funding sources.

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