Original Research

Clinicolaboratory Observations In Organophosphate Poisoning: Observations From A Tertiary Care Hospital

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Abstract

Background and aim: In some parts of the world, organophosphate poisoning is common and potential health risky and lethal. The clinic laboratory profile of people who have had organophosphate poisoning is very scanty. In the current study, an attempt is made to assess the clinic-laboratory profile of the patients confirmed to have incurred organophosphate poisoning and evaluate differences between healthy individuals with those who survived with those who succumbed to the poisoning. The aim of the study is to report the clinical and laboratory parameters in known cases of confirmed organophosphorus poisoning in a tertiary care hospital and compare the findings with healthy controls. Further, efforts are also made at comparing these parameters between individuals who survived versus succumbed to organophosphate poisoning. *Results:* A total of 73 known cases of Organophosphate poisoning from (2012- 2016) were studied. The majority of the cases were in the age group of 21-30 (30 %) and the majority of the cases 97% were suicidal in nature. The common symptoms observed were vomiting, abdominal pain, and diarrhea. The majority of the patients (75%) recovered, while 25% succumbed to the poisoning. There was a significant difference in the hematological, hepatic, and renal parameters between the control versus the poisoning group at the time of admission. *Conclusion:* The results indicate that a significant difference ($P < 0.05$ to 0.0001) in the clinical and laboratory parameters was observed between the controls versus the patient group as well as between the individuals who survived versus succumbed to the organophosphate poisoning.

Key words: Organophosphate poisoning, hematological, liver function tests, renal function tests.

Introduction:

Organophosphorus compounds are organic compounds that contains phosphorous element in it.[1,2] These compounds are primarily used in the agricultural fields as an insecticide and can be very toxic to man if consumed or if brought to contact.[1,2]

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Over the last few decades agricultural pesticides have become a common household item in rural areas of the developing world.[1,2] Due to their easy availability, they have also become commonly used for intentional self-poisoning.[1,2] Acute pesticide poisoning is now an important cause of morbidity and mortality worldwide[3]. World Health Organization (WHO) estimates published in 2005, indicate that around 3 million poisoning cases with 800,000 deaths occur annually due to poisoning and that about

99% of these deaths occur in the developing countries.[4]

Pesticide poisoning is a significant problem in India, and organophosphorus compounds are responsible for majority of the self-poisoning deaths in southern and central India.[5-8] Quantitatively, there are no reliable estimates in India as to how many people suffer from pesticide-related health effects every year and several reasons including lack of standardized case definition contributes to the lacunae in our scientific understanding. From a forensic view point, the case definition is inclusive of all circumstances of poisoning including suicide, homicide, non-intentional (accidental exposure) and occupational. In the farming areas of the developing world, organophosphate poisoning occur more likely as a suicide attempt than as an accident especially in adolescents and adults.[9] Pesticides used for self-poisoning include carbamates, organochlorines, and pyrethroids.[1] Poisoning can be caused from drinking, breathing in the vapors, or skin exposure of these compounds.[10]

The health effects associated with organophosphate poisoning are a result of inhibition of acetylcholinesterase (AChE) by organophosphorus compounds.[9] This then consequentially leads to buildup of acetylcholine (ACh) in different nerves and receptors in the body.[9] Pathologically, accumulation of ACh at motor nerves causes overstimulation of nicotinic expression at the neuromuscular junction. When there is an accumulation of ACh at autonomic ganglia synapses this causes overstimulation of muscarinic expression in the parasympathetic nervous system.[9] Mechanistically, organophosphates inhibit AChE, causing OP poisoning by phosphorylating the serine hydroxyl residue on AChE, which inactivates AChE.[9] AChE is critical for nerve function, so the irreversible blockage of this enzyme, which causes acetylcholine accumulation, results in muscle overstimulation[9].

From a clinical view point, the symptoms include increased saliva and tear production, diarrhoea, vomiting, small pupils, sweating, muscle tremors, and confusion.[9] Onset of symptoms often occurs within minutes to hours, but sometimes symptoms can take weeks to appear[11,12] and can last for days to weeks depending on the dose and potency of the ingested drug and general health of the individual.[9] In most cases the acute complications can include symptoms like fits, bradycardia, and delayed complications like paraplegia, sensory loss of limbs.[13]

From a therapeutic perspective, at times the medical management is difficult because there is little evidence with which to determine the best strategies for treatment and there is often intermittent supply of antidotes.[2,14,15] Treatment includes resuscitation of patients and providing oxygen, a muscarinic antagonist (usually atropine), fluids, and an acetylcholinesterase reactivator (an oxime that reactivates acetylcholinesterase by removal of the phosphate group).[15] Gastric decontamination is usually considered only after the patient has been fully resuscitated and stabilized.[15] Because of intermediate syndrome, the patient needs to be carefully observed after stabilization for changes in atropine needs, worsening respiratory function, and recurrent cholinergic features occurring with fat-soluble organophosphorus.[9]

Material and methods

This was a retrospective study, conducted at Department of Forensic Medicine. The study was undertaken following approval by the institutional ethics committee. A total of 73 patients who got admitted with the confirmed history of poisoning with Organophosphorus compound during the study period (2013 to 2016), were included in the study. Only known cases of poisoning with organophosphorus compounds were included in the study.

Patients with ambiguous history of organophosphate poisoning were excluded from the study. All the clinical, laboratory profile and treatment details during the study time period were considered. The data from individual patients satisfying the inclusion and exclusion criteria were noted down from individual files and entered into the Microsoft Excel. The demographic details were categorized into frequency, while the hematological and biochemical data were calculated to obtain mean±standard deviation (SD). All these details are represented in the tables. For comparison, results were compared as healthy individuals and Organophosphate poisoning patients and subjected to the Student -test. A value of 0.05 was considered significant. Similarly a comparison as done in clinical parameters observed in the living patients treated for organophosphorus poisoning and the patients who died as a result of Organophosphate poisoning

Results

Majority of the study population was male (64%) in comparison to female (36%). Most of the patients were in the age group of 21-30 (30%), followed by 31-40 (23%) and 41-50 (22%) (Table 1). The poisoning was found to be rare in the extremes of age groups. Most of the patients were of rural domiciliary origin (64%), compared to 36% from urban areas The bulk of the poisoning was suicidal in nature (97%), followed by a minority of accidental poisoning (3%) of the studied cases (Table 1). Most common symptom observed in the patients were vomiting (60%), abdominal pain (50%) and diarrhea (30%) (Table 1). The minority of the patients had lacrimation (12%) and salivation (10%). All the cases were treated using the antidote Atropine with symptomatic treatment to control gastrointestinal distress. 75% of the patients recovered from the poisoning, whereas 25% succumbed to death. It was observed at only 11 percent of the surviving cases suffered from comorbid conditions, in contrast to 39% of the dead individuals who were suffering from known comorbid conditions The hematological parameters studied (compared with control subjects) showed significant difference in Hemoglobin, total and differential count, ESR and platelet count (table 2). The hepatorenal lab findings showed statistically significant difference in the liver enzymes, blood urea and creatinine only (table 2). In comparison of living and dead individuals, statistically significant differences in the hematological and hepato-renal parameters were not profound except in the eosinophils seen in the differential leucocyte count, blood urea and total albumin count (Table 3). With regard to the treatment all were administered atropine, while pralidoxime was given to 30.14% of the patients (Table 4; Figure 1). The results also showed that ondansetron (41.09%), pantoprazole (42.47%), ranitidine (2.74%) and paracetamol were administered.

Among the 73 patients included in the study, 75% responded to the treatment. All patients required admission into the medical intensive care unit (ICU) for observation and for further management. 62 patients received gastric lavage on arrival to the emergency department. A fraction of the patients required intubation and mechanical ventilation from the emergency department itself. In the ICU, all patients received oxygen supplementation via nasal prongs, face masks and high flow nasal oxygenation. 26 patients with respiratory distress due to neuromuscular weakness and excessive secretions required intubation and mechanical ventilation.

History about the type of OP compound was sought from the patient's bystanders and based on the time since consumption of the compound; decision was taken to start the patient on infusions pralidoxime and atropine or atropine alone. For dimethyl compounds 6 hours was taken as the cut off and for diethyl compounds, upto 48 hours

since poisoning was considered the cutoff for oximes to be initiated. Dryness of the axilla was considered as the end point for stopping atropine therapy. All patients received hemodynamic monitoring with ECG, non-invasive blood pressure monitoring. Patients were tested hourly for worsening in muscle power. Assessment for bulbar weakness was also noted with patient's ability to swallow, cough and vocalize. Patient's oxygenation was monitored with pulse oximetry. Central venous catheters were inserted to patients requiring inotropic support and multiple intravenous infusions. Invasive blood pressure monitoring was done for 22 patients who were hemodynamically unstable requiring high dose vasopressors. All ventilated patients were started on a broad spectrum antibiotic (injection ceftriaxone 1gm IV thrice daily) and specific antibiotic changes were made based on clinical profile and microbiological culture sensitivity reports. Nutrition was taken care of by continuous nasogastric tube feeds in all ventilated patients and few patients with swallowing difficulty.

26 patients among the study group required mechanical ventilation in view of respiratory distress and micro aspirations. Daily sedation vacations were given for ventilated patients to look for improvement in muscle power and spontaneous breathing trials were given to assess readiness for extubation. Among those intubated, 11 patients died due to complications associated with prolonged ventilation and hemodynamic instability like ventilator associated pneumonia, multiorgan dysfunction, acute kidney injury, septic shock. Among these patients, 9 patients could be weaned off ventilatory support and extubated by day 7. Nine patients required prolonged ventilation (>7 days) and underwent tracheostomy in view of difficult weaning. Seven patients among those tracheostomized were decannulated after 21 days. Six patients among those intubated took discharge against medical advice.

Discussion

Organophosphate poisoning is one of the common types of cases admitted to the hospital and the ICU's in the developing world.[16,17] Easy availability of these compounds in the market has led to increased instances of intentional harm using these compounds.[18] In our study, male preponderance was observed in the present study, which was similar to previous studies.[19-21] The age group involved in the organophasphate was generally in the age group of 21- 40 (53%) which was similar to previous studies.[19,22] The people in the age group of 21-40 tend to be more prone for emotional conflict and hence more prone to self-harm, which was aptly demonstrated by our study. In the study, 97% of the organophosphate poisoning cases were suicidal in nature, which was consistent with previous studies from India,[19,22] Pakistan[23] and Turkey.[24] This is probably because of the easy availability of the compound in developing countries, which tend to be agricultural economies. However, the present study showed accidental exposure in pediatric age groups, which was similar to a study conducted in Egypt.[25] Our study shows gastrointestinal distress was a common sign and symptom in the poisoned patients in the form of vomiting, abdominal pain, diarrrhoea, lacrimation and in agreement to earlier studies.[19,22,25,26] The probable reason could be that the patients perceive gastrointestinal distress to be more agonizing in comparison to other symptoms. All the cases received the primary antidote Atropine, which was the primary method of treatment after stabilization of the patient and in agreement to previous observations.[9,24]

With regard to hematological indices, leukocytosis was the most common and in agreement to earlier reports.[27,29] In clinics, the complete blood count is a very vital endpoint and observations with 90

individuals who have had organophosphorus poisoning the WBC and neutrophil counts were important indicators and that a significant difference was seen between the severe poisoning group and the moderate poisoning group.[30] The most important aspect was that the receiver operating characteristic curve (ROC) graph, which indicates the clinical sensitivity and specificity for cut-off were 0.673 and 0.669 for WBC and neutrophil counts and that these were higher than that for cholinesterase (0.669) indicating complete blood count had clinical significance in organophosphorus poisoning.[30] Studies have shown that organophosphorus poisoning to cause oxidative damage, decrease in hemoglobin and to increase the total leukocyte counts.[31] Studies with agricultural workers occupationally exposed to organophosphate pesticides have also shown that the blood indices like RBC, WBC, monocytes, neutrophils, MCV, MCH, MCHC and platelet count were significantly altered when compared to controls.[16]

Chronic organophosphorus poisoning is shown to cause aplastic anemia, agranulocytosis, neutropenia, and thrombopenia in agricultural workers involved in spraying organophosphate pesticides.[16] Additionally, exposure to organophosphorus poisoning is shown to increase the risk of several hormonally-related cancers like that of breast, thyroid, and ovary among spouses of pesticide applicators.[32] To corroborate these observations, organophosphorus is also shown to induce genetic damage (as measured by quantifying the levels of micronucleus, nucleoplasmic bridges, apoptotic and necrotic cell frequencies) in the lymphocytes of people exposed to acute poisoning indicating its mutagenicity.[33] Cumulatively all these observations indicate the hematological toxicity of organophosphorus and also that they may have a prognostic role in casualty and medical care.

In addition to the hematological system, organophosphorus poisoning is also shown to affect the vital organs like kidney and liver. Kidney injury was observed to be a common phenomenon in our study and is in agreement to earlier observations.[34,35] Previous reports suggest that a transient elevation of the liver enzymes and acute kidney injury are observed in organophosphorus poisoning.[19,36,37] Hypokalemia, hyperglycemia, acute renal failure, transient elevation of liver enzymes can occur in organophosphorus poisoning.[36] Further Panda and coworkers.[38] have observed altered renal function between the survivors and non survivors suggesting their importance in predicting mortality. The transient renal injury may be due to both a direct action of the organophosphate, causing tubular cell necrosis or secondary mechanism that followed the cholinergic crisis, causing hypovolemic shock and rhabdomyolysis. Seminal studies by Lee and co workers[34,35] have documented that bradycardia, dehydration with hypovolemia, and hypotension that induced renal hypoperfusion, direct toxic effects on renal parenchyma and convolutions, endothelial cell damage, activation of immune and inflammatory responses, formation of free radicals, convulsive seizure, and muscular fasciculation–related rhabdomyolysis may all contribute to the decline of renal function after acute poisoning from organophosphate.[34,35]

With regard to hepatic parameters, in our study, hypokalemia and transient elevation of liver enzymes were found in 15.03% and 13.5 % of the cases respectively. A significant increase in the liver enzymes, particularly the ALP and AST was noted compared to the control group and this observation is consistent with previous studies.[35,39-41] Previous reports have shown that liver injury was seen in 9.8% and 5.17% of cases and control group, respectively in OP poisoning and mortality was higher in cases than

controls (22.5% vs 6.32%).[37] Reports suggest that the liver enzymes are elevated in people chronically exposed to OP poisoning. In our study when compared to patients who survived, the liver enzymes were found to be higher among the patients who succumbed to the effects of the poison indicating that the deranged, liver and renal parameters, needs to be critically observed during admission for care. To substantiate these, studies have shown that the prevalence of liver and kidney dysfunction was observed to be high in occupationally exposed farm workers and plant protection agents from Burkina Faso [42].

From a pharmacological view point, the clinical effects of organophosphate poisoning are due to the stimulation of the muscarinic and nicotinic receptors. The stimulation of muscarinic causes symptoms such as diarrhoea, urination, miosis, bradycardia, bronchorrhea, bronchospasm, emesis, lacrimation, and salivation; while triggering the nicotinic receptors in the sympathetic ganglia and neuromuscular junction leads to mydriasis, tachycardia, weakness, hypertension, and fasciculations [43]. The "mixed" or concomitant nicotinic and muscarinic clinical effects can be perplexing at times and can lead to misdiagnosis[44]. Clinically the CNS effects are observed to be diverse and worse, can be both nonspecific and severe [45]. Of all the signs muscle weakness and paralysis is of particular importance, as this can contribute to respiratory arrest and lead to death of the individual. Development of diffuse weakness that is termed as "Intermediate syndrome" often leads to respiratory failure and requires ventilatory assistance. The immediate and efficient management at this phase of the illness is vital at improving the overall recovery rates [46].

From a therapeutic perspective, removal from the source and decontamination of the patient is the first and vital step in critical care of an individual with OP poisoning and is generally done before arrival to the

Table 1 Details of the patients

Age Distribution of all patients	0-10	1
	11-20	3
	21-30	22
	31-40	17
	41-50	16
	51-60	9
	61-70	2
	71-80	2
	81-90	1`
	Total	73
Sex distribution of all patients	Male	47
	Female	26
	Total	73
Manner of death	Suicidal	71
	Accidental	2
	Homicidal	0
	Total	73
Mortality	Survived	55
	Death	18
	Total	73
Signs and symptoms in patients	Vomiting	60%
	Abdominal Pain	50%
	Diarrhea	30%
	Lacrimation	12%
	Salivation	10%

Heath care facility [47]. Further decontamination is warranted only after initial stabilization and injury assessment [48]. In most cases loss of airway and respiratory drive, and seizures are the most common cause for mortality. In lieu of these observations early control of airway and breathing is often required and may need to be performed concurrently with decontamination in patients of OP poisoning. To achieve this rapid atropinization should be initiated even before oxygen administration because oxygenation may be impossible until secretions are controlled[2,49]. In our observation all were administered atropine, (Table 4). Atropinization, which has been used for over half a century still remains the mainstay in the clinical management of all patients with OP poisoning and significantly reduces the morbidity and mortality. Pralidoxime was given to 30.14% of the patients (Table 4). Reports indicate of pralidoxime is limited to only certain cases and various factors need to be considered before it's use [50]

Conclusion

Organophosphate poisoning is common in developing countries on account of easy availability of these compounds and because of the agrarian nature of these economies. The epidemiology of poisoning is well studied in different centers around the globe; however there is a considerable dearth of studies correlating the laboratory parameters to organophosphate poisoning and our study hopes to throw light on these findings. The present study postulates the need to monitor the clinical signs and symptoms and the associated hematological, hepatic and renal parameters for the better prognosis of the patient and limit the mortality in these patients. Further multi-centric studies may be required to gather more data which will help in further studying this particular component of Organophosphate poisoning.

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Table2 : Comparison of Clinical and lab parameters between the OP poisoning patients and control

OP	Control	OP poisoning	t	Df	P
Hb	12.5±1.06 (9-15.4)	14.08±2.2 (8.1-18.3)	-5.63	85.73	<0.0001
TC	8201.61±1729.14 (5100-13600)	15283.82±9220.72 (3300-71800)	-6.27	69.59	<0.0001
N	58.31±8.29 (36-82)	76.78±15.54 (26-94)	-9.17	89.99	<0.0001
L	35.9±9.16 (14-54)	16±12.76 (3-52)	11.42	107.64	<0.0001
E	3.66±2.15 (1-9)	1.93±2.66 (0-20)	4.64	117.8	<0.0001
M	2.53±1.74 (1-14)	5.29±2.28 (0-12)	-8.75	112.66	<0.0001
ESR	5.39±1.88 (2-9)	13.22±19.76 (2-90)	-2.66	44.29	0.005
Platelets	214853.2±85991.37 (73000-388000)	249515.2±74629.18 (55000-471000)	-2.81	152.28	0.003
AST	23.19±8.49 (10-60)	67.78±179.35 (14-1477)	-2.03	66.16	0.02
ALT	21.59±12.4 (10-83)	43.52±96.09 (10-790)	-1.89	69.26	0.03
ALP	73.1±19.03 (38-108)	89.14±41.81 (16-289)	-2.95	79.96	0.002
T. Protein	7.3±0.42 (6.4-8.2)	7.24±1.03 (4.54-9.64)	0.45	75.57	0.32
Albumin	4.3±0.43 (3.12-5.07)	4.4±0.62 (2.69-5.76)	-1.22	97.72	0.11
Globulin	2.98±0.33 (2.4-3.5)	2.94±0.62 (1.44-4.9)	0.52	84.29	0.30
A/G Ratio	1.46±0.26 (0.9-1.9)	1.53±0.39 (0-2.47)	-1.26	94.49	0.10
T. Bil	0.6±0.14 (0.2-0.86)	0.85±0.9 (0.17-5.82)	-2.41	69.09	0.009
CB	0.32±0.11 (0.1-0.6)	0.38±0.73 (0.1-5.23)	-0.69	67.74	0.24
UB	0.27±0.08 (0.1-0.6)	0.48±0.42 (0.07-2.3)	-4.06	67.96	<0.0001
Na	139.01±1.85 (136-143.8)	138.62±4.39 (127-147)	0.69	81.67	0.24
K	4.29±0.38 (3.6-5.07)	3.71±0.51 (2.78-5.18)	8.23	111.8	<0.0001
Cl	101.54±2.2 (95.8-106)	96.7±11.92 (12.9-110)	3.24	67.67	0.0009
Urea	20.97±8.04 (10-40)	24.34±12.82 (10-76)	-1.97	96.55	0.026
Creatinine	0.75±0.2 (0.49-1.35)	1.27±2.09 (0.17-18)	-2.08	68.68	0.02

Table3 : Comparison of Clinical and lab parameters between survived and expired patients of OP poisoning

OP	Alive (excluding LAMA)	Dead	t	df	P
D in hospital	8.09±3.78 (1-22)	3.53±5.23 (1-23)	3.29	22.45	0
D in MICU	2.25±2.07 (1-7)	1.6±0.97 (1-4)			0
D on ventilator	1±0 (1-1)	1±0 (1-1)	NaN	6	0
BP sys	122.68±15.73 (100-190)	123.33±36.46 (90-240)	-0.07	19.47	0.47
BP dia	79.79±10.93 (60-110)	76.67±16.45 (50-120)	0.74	22.99	0.23
RR	18.86±4.31 (14-36)	21.53±6.75 (6-30)	-1.43	18.25	0.08
HCO3	19.93±4.22 (12.5-28.5)	19.21±6.46 (11.5-28.4)	0.27	8.48	0.4
pH	7.32±0.16 (6.8-7.47)	7.3±0.34 (6.8-7.54)	0.09	3.34	0.47
PTT	27.55±4.82 (14.7-38.6)	24.05±2.9 (22-26.1)			0
INR	1.04±0.13 (0.86-1.5)	1.97±2.73 (0.89-10.1)	-1.13	10.02	0.14
Pulse	88.51±19.51 (46-140)	103.39±25.94 (58-180)	-2.18	25.82	0.02
PO2	104.31±66.87 (17-315)	123.5±145.83 (17-376)	-0.31	5.81	0.38
PCO2	38.18±11.59 (29-79)	58.67±25.3 (39-100)	-1.91	5.81	0.05
Hb	14.34±2.19 (8.1-18.3)	13.96±2.04 (10.5-17.8)	0.63	27.59	0.27
N	75.7±16.33 (26-94)	79.94±11.22 (60-92)	-1.15	37.99	0.13
L	16.7±12.99 (3-52)	14.13±10.56 (3-33)	0.79	31.69	0.22
E	2.28±3.14 (0-20)	1.06±0.25 (1-2)	2.63	47.68	0.01
M	5.34±2.26 (1-11)	4.81±2.4 (0-12)	0.77	24.67	0.22
ESR	13.1±19.56 (2-90)	8.27±3.98 (2-15)	1.3	35.93	0.1
TC	15680.43±10640.27 (3300-71800)	15606.25±5404.13 (3700-24100)	0.04	51.51	0.49
Platelet	248934.8±64294.6 (110000-436000)	238066.7±93621.93 (55000-370000)	0.42	18.5	0.34
Urea	22.72±10.95 (10-76)	32.31±16.14 (18-74)	-2.21	20.01	0.02
Creat	1.31±2.52 (0.17-18)	1.32±0.49 (0.69-2.54)	-0.03	54.85	0.49
Tbil	0.76±0.57 (0.17-2.7)	1.26±1.53 (0.28-5.82)	-1.28	16.48	0.11
CB	0.26±0.16 (0.1-0.81)	0.78±1.43 (0.12-5.23)	-1.46	15.13	0.08
UB	0.5±0.46 (0.07-2.3)	0.48±0.32 (0.14-1.45)	0.18	34.8	0.43
TP	7.35±1.03 (4.64-9.64)	6.89±1.12 (4.54-8.21)	1.44	25.15	0.08
Albu	4.52±0.54 (3.13-5.49)	4.17±0.77 (2.69-5.76)	1.69	20.82	0.05
Glob	2.97±0.62 (2-4.9)	2.73±0.62 (1.44-3.6)	1.28	24	0.11
AST	40.2±33.87 (14-169)	152.67±369.2 (19-1477)	-1.18	14.08	0.13
ALT	31.91±32.77 (10-167)	79.5±190.63 (10-790)	-0.99	15.3	0.17
ALP	87.69±37.08 (16-186)	91.2±60.14 (16-289)	-0.21	17.68	0.42
Na	138.66±3.91 (127-146)	138.5±5.97 (127-147)	0.1	19.58	0.46
K	3.61±0.42 (2.78-4.45)	3.92±0.67 (2.83-5.18)	-1.73	19.22	0.05
Cl	97.98±5 (86.7-109.7)	97.49±7.19 (81.7-110)	0.25	20.4	0.4