

Interventions for the Treatment of Organophosphorus Pesticide Poisoning

Self-poisoning with Organophosphorus (OP) pesticides is a major problem across the Asia Pacific region. They cause the majority of poisoning deaths in India, except in some northern areas where aluminium phosphide predominates. There is no effective treatment for aluminium phosphide poisoning; in contrast, moderately effective treatments and antidotes exist for OP poisoning which if used well can reduce mortality. However, the evidence for most interventions is weak and more effective therapies are required if the case fatality ratio is ever to be consistently reduced to below 10%.

PATHOPHYSIOLOGY

The effect of OP pesticides is due to the inhibition of acetylcholinesterase (AChE) in synaptic clefts of the neuromuscular junction, autonomic nervous system, and CNS. Acetylcholine is no longer broken down, resulting in sustained stimulation of post-synaptic receptors and the classic picture of OP poisoning described in every textbook. Deaths occur acutely due to respiratory failure or cardiovascular collapse,

and later due to peripheral respiratory failure and the complications of aspiration and long term ventilation.

RESUSCITATION

Effective and rapid resuscitation of patients with stabilisation of the airway is essential for a good outcome. Ventilation will be required for a significant number of patients.



ATROPINE

Administration of atropine is fundamental to the management of OP poisoning. Atropine antagonises acetylcholine's effects on muscarinic receptors and treats the early parasympathetic features of OP poisoning, increasing the heart rate and blood pressure and reducing excess fluid and bronchospasm in the lungs.

Stabilisation of the patient requires rapid administration of intravenous atropine to improve cardiac and respiratory function – to 'atropinise' the patient. Although this has been standard practice for many years, the ideal regimen is still not known. A review of textbook recommendations on patient atropinisation found 35 different regimens. Most were not specific, suggesting doses such as 1-5mg, every 5-20 minutes; some

would have required several hours to give 25mg.

The fastest method to atropinise patients is to give a 1-3mg initial bolus of atropine and to see whether it reverses sweating, bradycardia, hypotension, bronchospasm, and bronchorrhoea. If there is no response at five minutes, the dose is doubled and doubled again until there is a clear improvement in the patient's condition – using very large boluses if necessary. This method is preferred since rapid stabilisation of cardiorespiratory function is required and it results in the fastest administration of atropine among the regimens reviewed.

There has been only one published controlled trial of atropine. It found that boluses followed by an infusion were more effective than boluses alone; however, the study may have over estimated the benefits as it used historical control patients. In the absence of better evidence, the World Health Organization (WHO) currently recommends the use of boluses followed by an infusion to keep patients atropinised.

OXYGEN AND IV FLUIDS

Textbooks often state that atropine should not be given until the patient has been given oxygen, because of a risk of inducing ventricular dysrhythmias. However, the evidence for this is weak and atropine can be given routinely to patients before oxygen is available without inducing dysrhythmias. While it is preferable to give oxygen to OP

This issue has attempted to cover three different areas of poisoning – chemicals, medicines & bites that are encountered by patients, doctors and health workers frequently. The articles are contributed by eminent persons or institutions and the editorial team thankfully acknowledges their efforts.

poisoned patients, the absence of oxygen should not prevent the urgent administration of atropine in small rural hospitals since atropine will improve patient oxygenation by reducing bronchospasm and bronchorrhoea, and increasing the heart rate.

The increased secretions found in OP poisoning produce intravascular volume depletion. Administration of 500-1000ml of normal saline in a severely poisoned patient should be considered.

PRALIDOXIME

The effectiveness of oximes such as pralidoxime is unknown. However, the WHO currently recommends a loading dose of at least 30mg/kg given over 20-30 minutes followed by an infusion of at least 8mg/kg/hr. Too fast administration of the bolus induces vomiting that risks aspiration. A randomised controlled trial (RCT) is now underway in Sri Lanka to test the effectiveness of this regimen.

An RCT carried out at the Christian Medical College, Vellore, reported increased incidence of respiratory failure and death in patients who received 3-4g of pralidoxime/day as an infusion, but no loading dose. This result appears to contrast with clinical

studies carried out in Germany and may be explained in part by the long interval between poisoning and hospital admission in the study (median 12hrs). This long delay would have had two consequences: changes in the inhibited AChE ('ageing') in patients taking dimethyl OPs such that pralidoxime could not have worked, and an opportunity for complications such as aspiration or anoxia to have occurred before admission (patients may have died of these complications which would not have been affected by oximes).

Some OP poisoned patients are unlikely to benefit from oxime therapy – for example those with severe complications from the pre-hospital period, those presenting after 12 hours with dimethyl OP poisoning, and those with very severe poisoning in which high OP blood concentrations simply re-inhibit the AChE that the pralidoxime has just reactivated.

More recent studies also suggest that S-alkyl OPs such as metamidophos and profenofos age very quickly and require oxime therapy to be started within minutes, rather than hours. In the context of the rural Asia, this is not practical and such OPs may be best considered as not responding to pralidoxime. Despite these caveats, it is probably best at the moment to treat all patients with OP poisoning

with pralidoxime until definitive studies, with identification of OPs, are reported.

GASTRIC DECONTAMINATION

Gastric decontamination is unlikely to benefit most patients as OPs are rapidly absorbed. Thus, gastric lavage or activated charcoal

should not even be considered until the patient has been resuscitated and stabilised with administration of atropine, fluid, and oximes. In patients who have not received atropine, the passage of a large orogastric tube risks inducing vagal responses that can result in asystolic cardiac arrest. Lavage in a struggling patient also has a high risk of causing pulmonary aspiration of the pesticide and its solvent. Thus lavage should only ever be performed in a cooperative or intubated patient.

Forced emesis by any means is not recommended since patients may rapidly lose consciousness and their airway control while vomiting, risking aspiration. Preliminary results of a large RCT of activated charcoal performed in Sri Lanka showed no effect of superactivated charcoal in OP poisoning. However, until this study is formally analysed and reported, it seems reasonable to give a single dose of charcoal to all patients presenting with 2 hours.

OTHER DRUGS

Diazepam is used for the treatment of OP induced seizures, and can also be used to reduce agitation. In the absence of good information about the level of brain damage in poisoned patients, it is not yet possible to determine the role of routine use of CNS sedatives such as NMDA blockers or diazepam. Bicarbonate is used in Iran and Brasil for OP poisoning but clinical studies have not shown benefit.

FUTURE TREATMENTS

The current regimen of resuscitation, oxygen, atropine, oximes and diazepam is only partly effective, with case fatality ratio over 10% being common in many hospitals. There are many other possible interventions that need to be taken through clinical development and, if effective, provided at an affordable cost.

References for this information can be found online at <http://ccforum.com/content/8/6/R391>

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Down To Earth

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Drug Poisoning

INTRODUCTION

Poisoning by drug overdose constitutes a large fraction of intentional suicidal deaths around the world. In a few cases it may be accidental or homicidal. Whatever the case may be, if the victim receives prompt medical attention and good supportive care, the prognosis is a good one. The following is an overview of the various mechanisms by which drug overdose can harm the body, followed by an account of preliminary and specific measures that can be taken in a case presenting with a drug overdose.

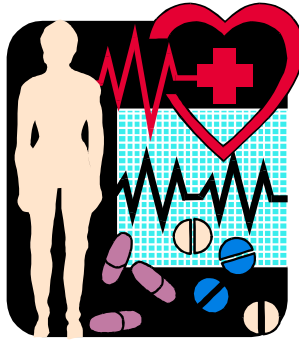
HOW DOES A POISONED PATIENT DIE?

An understanding of the common mechanisms of death due to poisoning by drugs can help doctors in diagnosis and management of the patient. The various ways in which the different systems are affected in drug poisoning are mentioned here.

The Central Nervous System:

(CNS) depressant drugs not only impair the consciousness and lead to coma but they may also lead to a loss of protective reflexes and respiratory drive of the patient. Cause of death here can be an airway obstruction by flaccid tongue, aspiration of gastric contents into the tracheobronchial tree or respiratory arrest. These are seen in an overdose of *narcotics, barbiturates, alcohol* and other *sedative-hypnotic drugs*. Seizures can be a cause of death by causing pulmonary aspiration, hypoxia and brain damage. These often result from overdose of *amphetamine, cocaine, antipsychotic, anti depressants, diphenhydramine, theophylline; isoniazid* etc. These drugs can also cause muscular hyperactivity and rigidity, which may in turn, lead to complications of hyperthermia, muscle breakdown, myoglobinuria, renal failure, lactic acidosis and hyperkalemia.

The Cardiovascular System: Overdose of cardio active drugs such as *ephedrine, amphetamine, cocaine, tricyclic antidepressants, digitalis and*



theophylline, can cause death by altering the cardiac contractility, lead to hypotension, or cause arrhythmias. Hypotension may be caused by depression of cardiac contractility; peripheral vascular collapse is due to blockade of α -adrenoceptor mediated vascular tone; hypovolemia maybe due to vomiting, diarrhoea, fluid sequestration or due to temperature deregulating effects leading to hypothermia or hyperthermia. Arrhythmias like ventricular tachycardia or fibrillation, commonly caused by these drugs, may also prove to be lethal.

Cellular Hypoxia: Drugs that interfere with the transport or utilization of oxygen may cause cellular hypoxia in spite of adequate ventilation and oxygen administration. These drugs such as *cyanide* clinically present with tachycardia, hypotension, severe lactic acidosis and signs of ischemia on ECG.

Other Organ System Damage may occur due to drug poisoning but may be delayed in onset. A few examples are that of *paraquat* affecting lung tissue, resulting in pulmonary fibrosis, which begins several days after ingestion. A similar example is that of massive hepatic necrosis due to poisoning by *paracetamol*, which may result in hepatic encephalopathy and in death two to three days later after ingestion.

Certain **Behavioural Effects of Drugs** may be a cause of death in an indirect manner. For example, *alcohol* or *sedative-hypnotic* drugs can cause motor vehicle accidents; patients under the influence of hallucinogens like *phencyclidine* or *LSD* may die in fights or falls from high places.

MANAGEMENT OF THE POISONED PATIENT

Initial Management

The initial management of a patient with coma, seizures or otherwise altered mental status should first aim at restoring the Airway, Breathing and Circulation (ABC), like in any other case. Specific toxicological diagnosis and management should follow later but as soon as possible.

Once the ABCs are maintained, then the cause of an alteration in mental status should be further probed into. Hypoglycemia, a much more common finding in patients, also presents itself simulating intoxication. Therefore, a dextrose infusion, 50 ml of 50 % D, IV, should be given promptly. Alcoholics and malnourished are also given 100mg of thiamine, along with dextrose, to prevent Wernickes' encephalopathy. In case a drug overdose is suspected, *naloxone*, an opioid antagonist, can be given in a dose of 0.4-2mg IV, to reverse respiratory and CNS depression due to all varieties of *opioids*.

Toxicological Diagnosis

To proceed for making a toxicological diagnosis for the patient, the history, physical findings and lab results have to be noted.

SUMMARY

- Monitor the airway (recovery position +/- intubation)
- Maintain normoxia (+/- IPPV)
- Maintain body temperature
- Correct any hypotension (+/- volume expansion +/- renal dilators) or hypertension
- Correct acid-base or electrolyte disturbance
- Treat any fits (Diazepam)
- Monitor for dysrhythmias (secondary role for antiarrhythmics)
- Beware of skin blistering and rhabdomyolysis
- Remember to take account of concurrent medical problems.

Oral statements obtained as **history**, should be substantiated with any articles recovered from the patient's side, like empty bottles, vials, syringes or drug strips, in order to quantify the drug ingestion.

Clinical Examination

Certain clues from the **physical examination** of the patient can help in arriving at the diagnosis. A few examples are as follows:

1) Vital signs:

a) **Blood pressure** – Hypertension and tachycardia are typical with *amphetamines, cocaine and anti-cholinergic drugs*.

Hypotension and bradycardia are characteristic features of overdose with *calcium channel blockers, β -blockers, clonidine and sedative-hypnotics*. Hypotension and tachycardia is common with tricyclic *anti-depressants, phenothiazines, vasodilators and theophylline*.

b) **Respiration** – Rapid respiration is typical of *salicylates, carbon monoxide* and others producing metabolic acidosis or cellular hypoxia.

c) **Temperature** – Hyperthermia is associated with *sympathomimetics, anticholinergics, salicylates* and drugs producing seizures and muscular rigidity. Hypothermia can be produced by CNS depressants.

2) **Eyes: Miosis** – constriction of pupils, is typical of *opioids, clonidine, phenothiazines, cholinesterase inhibitors* and *sedative* drugs.

Mydriasis- dilatation of pupils is common with *amphetamines, LSD, atropine and other anticholinergic agents*.

Horizontal nystagmus is characteristic of intoxication with *alcohol, phenytoin, barbiturates* and other *sedatives*. Presence of both vertical and horizontal nystagmus is suggestive of poisoning with *phencyclidine*.

3) **Skin**: It may appear flushed, hot and dry in poisoning with *atropine* and other *antimuscarinics*. Excessive sweating occurs with *sympathomimetics, nicotine and anti cholinesterase agents*. Cyanosis may be due to hypoxemia or methemoglobinemia. Icterus may suggest hepatic necrosis due to *paracetamol* toxicity.

4) **Nervous system**: Nystagmus, dysarthria and ataxia are typical of

phenytoin, carbamazepine, alcohol and other sedative intoxication. Twitching and muscular hyperactivity are common with *atropine, cocaine and other sympathomimetic* agents. Muscular rigidity is seen under effect of *haloperidol* and other *anti psychotics*.

Laboratory Tests

Laboratory tests usually required to arrive at a diagnosis or for confirmation are ABG – Arterial Blood Gas, serum electrolytes, serum osmolality, renal function tests and ECG.

Drugs that may induce an elevated anion gap acidosis are:

Type of elevation of the Anion gap	Agents
Organic acid metabolites	Methanol, ethylene glycol
Lactic acidosis	Cyanide, carbon monoxide, ibuprofen, isoniazid, metformin, salicylates, valproic acid, any drug induced seizures, hypoxia or hypotension

Drugs that cause alteration in serum potassium levels are:

Drugs that may cause hyperkalemia	Potassium itself, β -blockers, digitalis glycosides, potassium sparing diuretics and fluoride
Drugs that may cause Hypokalemia	Barium, β antagonists, caffeine, theophylline, thiazide and loop diuretics

Drugs that cause alteration in serum potassium levels are; hazardous as they may lead to cardiac arrhythmias. Clinical examination and laboratory tests are usually sufficient to generate a tentative diagnosis and an appropriate treatment plan.

Decontamination

It involves removing toxins from the skin or gastrointestinal tract. Emesis with *Ipecac* syrup or gastric lavage with *normal saline* is generally used if the treatment is initiated within an hour of ingestion of the drug. **Activated charcoal** is useful in binding the ingested drugs and reduces their absorption. It should be given in a ratio of 10: 1 to estimated dose of drug by weight (dose 50-100g; surface area 1000m²/g) Effective-

ness may be prolonged if gastric emptying is delayed by the drugs ingested (e.g. *opiates or anticholinergics*), by the formation of tablet bezoars (e.g. *salicylates*), ingestion of sustained-release preparations (e.g. *theophylline*).

Specific Antidotes

Selective antidotes are available only for a few classes of drugs.

Drug	Antidote
Coumarin warfarin	Vitamin K
Haloperidol	Benz-tropine
Heparin	Protamine sulphate
Benzodiazepine	Flumazenil
Opiates	Naloxone
Paracetamol	N-Acetyl cysteine

METHODS OF ENHANCING ELIMINATION OF DRUGS

1) Dialysis procedures:

a) Peritoneal dialysis – not very effective in removal of drugs

b) Hemodialysis – helps in removal of drugs, its toxic metabolites, if any, and correction of fluid and electrolyte imbalance. It is especially useful in *salicylate* poisoning

c) Haemoperfusion – Blood is pumped from the patient in a venous catheter through a column of adsorbent material and then recirculated to the patient. It removes many high molecular weight toxins (requires low Vd i.e. <51/kg). It can enhance whole body clearance of *salicylate, phenytoin, phenobarbital, theophylline, and carbamazepine*.

2) Forced Diuresis and urinary pH manipulation:

a) Alkalinisation of urine – (pH >7.5) is useful in cases of *salicylate* and *barbiturate* poisoning

Acidification of urine – urinary pH kept less than 7.0 – useful mainly in *amphetamine* poisoning

References:

- 1) Kent R Olson: Basic and Clinical Pharmacology, 9th ed. the McGraw-Hill companies 2001
- 2) Curtis D. Klaasen: Goodman & Gilman's: The Pharmacological Basis of therapeutics, 10th ed. The McGraw-Hill companies 2001

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BITES



SNAKE BITES

Of the 216 species of snakes found in India, 52 are poisonous. The common poisonous land snakes in India belong to families of Viperidae (vipers) and Elapidae (cobras). Snake venoms are complex mixture of enzymes, low molecular weight polypeptides, glycoproteins and metal ions which can adversely affect multiple systems and organs of the human body.

Management: Aims of treatment are to: (a) slow down absorption of venom from site of bite, (b) Neutralise venom as quickly as possible and (c) Prevent complications.

Field Management (First Aid): the objective is delivering the victim to a place where optimal medical care is available quickly and safely. The victim should be as inactive as possible to limit spread of the venom and the bitten part immobilised by using a splint. A tourniquet can be applied lightly about 10 cm above the bite to prevent lymphatic spread of the venom. The bandage should be released only after antsnake venom has been given.

Hospital Management: Vital signs of the patient should be assessed hourly. Large bore intravenous line in unaffected extremities should be started. Blood for laboratory investigations (haemogram, coagulation profile, kidney function profile, and cross) should be sent. Hypotension if present, is to be corrected by giving i.v. fluids, volume expanders, and dopamine as required. Fresh blood transfusion or fresh frozen plasma may be required to correct coagulopathy. Patients with respiratory paralysis need ventilatory support. All patients of snake bite should receive tetanus toxoid as snake venom contains clostridia and other bacterias. Antibiotics should be given if local infection is severe.

DOG BITES AND RABIES

Dogs are the most common vector of rabies virus for humans. Rabies is an acute viral infection that affects the central nervous system. Rabies can also, be transmitted by other animals like cat, monkey, mongoose, fox, wolf and bat. Rodents are rarely infected with rabies virus. The incubation period of rabies ranges from 7 days to over a year (mean 1 to 2 months). Rabies is fatal; the median period of survival is 4 days after onset of symptoms.

Treatment: Once symptoms of rabies develop, there is no cure. Aim of treatment is to alleviate the pain and suffering of the patient.

Post exposure prophylaxis: Post exposure prophylactic treatment must be started at the earliest, to ensure that the individual is protected before the virus infects the central nervous system. All unprovoked bites or where the animal cannot be traced or identified, should undergo post exposure prophylaxis of rabies, which includes local treatment of the wound and administration of antirabies immunoglobulin (serum) for passive immunisation together with antirabies vaccine (active immunisation).

Local wound treatment: The wound should be washed with plenty of water and soap to remove all traces of saliva. 1% to 4% benzalkonium chloride or 1% cetrimonium bromide may be applied as they inactivate the virus. Seventy per cent of alcohol or povidone iodine also gives good results. Tetanus toxoid should be administered. Stitching of the wound should be avoided and if unavoidable minimum stitches should be done.

Post-exposure vaccination: (a) **Passive Immunisation:** In severe (high risk) exposures 20iu/kg Human Rabies Immunoglobulin (HRIG) as a single dose, or if it is not available, Equine

antiserum 40iu/kg is given – 50% of the dose is infiltrated locally and the rest is administered intramuscularly into the gluteal region. (b) **Active Immunisation:** Currently the following rabies vaccines are available: i) Purified chick embryo cell rabies vaccine.

ii) Inactivated rabies vaccine obtained by culture vero cell lines
iii) Human diploid cell rabies
iv) Purified duck embryo rabies vaccine.

Immediately after exposure, a full course of treatment consisting of 5 injections on days 0 (day of exposure), 3, 7, 14 and 30, and a booster dose on day 90 should be given intramuscularly in the deltoid region.

Pre-exposure prophylaxis: Individuals at high risk exposure to rabies exposure are recommended to receive three cell cultured vaccine on days 0, 7 and 28.

INSECT BITES AND STINGS

Bees, wasp, hornet stings and ant bites are a common occurrence. Usually they cause minor discomfort and require only symptomatic treatment like, local application of calamine lotion to reduce irritation, systemic antihistaminics to reduce pruritus, and analgesic for pain. If severe symptoms are present systemic corticosteroids may be needed. Some individuals may develop anaphylactic reaction and they should be observed for atleast 24 hours along with treatment.

SCORPION STINGS

Of the approximately 1000 known species of scorpions only about 30 are potentially lethal. Following a scorpion sting, there may be pain, burning sensation, echymosis and oedema which subside within a few hours and can be managed by giving simple analgesics and antihistamines.

**Dr S Banerjee, Clinician
in a charitable clinic, Delhi**

Banned Drug

The Central Government of India has notified on December 13, 2004 the ban on Rofecoxib formulations for human use. It is in the context of certain risk to human beings. It prohibits manufacture, sale and distribution Rofecoxib with immediate effect. The notification implies immediate withdrawal of the stock that is still in the retail outlets and supply chain.

This follows the recommendation of the National Pharmacovigilance Advisory Committee meeting held on October 11, 2004 for banning the drug, in the context of Merck withdrawing the drug in September 2004.

ALERT Kerala Drugs Controller has banned the following batches of drugs for substandard quality

Sample tests conducted during the month of November 2004 at the Thiruvananthapuram drug control laboratory revealed a few batches of drugs, marketed by about a dozen companies in Kerala, were of substandard quality. They are:

Medicine	Company	Batch Number
Isosorbide Dinitrate tablets IP	Micron Pharmaceuticals, Vapi	T 05014
Absorbent cotton wool IP	Konark Surgicals Ltd, Nalgonda	383
Cloxacillin tablets IP	Modern Pharmaceuticals, Kandli	MC 014
Paracetamol tablets IP	Modern Pharmaceuticals, Kandli	MT 053, MT 054 and MT 190
Paracetamol tablets	Vyasa Drugs and Pharmaceuticals Ltd, Aalappuzha	PB 089, 091, 090
Paracetamol tablets	Omega Biotech Ltd., Ghaziabad	T 4081
Cotrimoxazol tablets IP	Pure Pharma Ltd, Indore	30044
Sparfloxacin	Sim Laboratories Ltd, Nagpur	tablets PE 44 of batch A 401
Prednisolone tablets IP	Civil Drugs Laboratories, Delhi	PR 01
Calcium lactose tablets IP	Unicure (India), Noida	CITT 20106
5 ml disposable syringes	EE Hand Syringes Pvt. Ltd., Palakkad	B-016
Aluminium Hydroxide tablets IP	Bharath Parenterals Ltd., Baroda	T-4090

The concerned manufacturers have been asked to withdraw the substandard batches from the market by the drug control department.

Compiled from Chronicle Pharmabiz

Cox2 Inhibitors

The National Pharmacovigilance Advisory Committee (NPAC) has asked drug companies which licensed to manufacture and sell generics of Cox-2 inhibitor category drugs to submit additional data on the product safety with immediate effect. The move is a fall out of the reported ADRs and the subsequent withdrawal of rofecoxib, a cox-2 inhibitor drug, from the international markets including India.

The manufacturers of COX-2 drugs like, celecoxib, valdecoxib parecoxib generics are expected to initiate additional studies such as safety profile during use for more than 18 months, potential reactions of the drug in various dosage forms and designs etc. immediately and submit the data to the NPAC.

'I just saw the July - September 2004 (issue 17) of the Rational Drugs. The tidbits of information on the drugs that have been registered in India and fake approvals are useful. I have sent the information to WHO for their counterfeit's database.'

Dr Krisantha Weerasuriya

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The focus theme for the next issue will be on 'Mental Health.' We welcome readers to contribute for 'Readers Forum' and Adverse Drug Reaction (ADR).

Rational Drugs

A Health Education Publication from the *Policy Advocacy & Research Group of CMAI.*



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Design & Production

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Published by

General Secretary, CMAI

All correspondence to:

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Plot No 2, A-3 Local Shopping Centre
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Printed at: Creative Lab