COMMUNICATION:

THE SECOND ASIAN CONGRESS OF PAEDIATRIC
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Theme: GROWING UP IN SOUTH EAST ASIA

1. Patterns of Growth and Development in Children in South East Asia
   (To promote family planning concepts through child care services, and research)

   Guest Speakers:


   2. PROF. R. KUSUMANTO SETYONEGORO. (Indonesia): "Concerning Some thought on perinatal development in Children in Indonesia."

   3. Prof. PRASONG TUCHINDA (Thailand): "Concerning The influence of socio economical factors, particularly the size and education of family, in promoting growth and development."

   4. Prof. NOBORU KOBAYASHI (Japan): "Concerning Change in the pattern of growth and development in children and family planning in Japan.

   5. Prof. MCDONAL (Australia): "Concerning Growth and Development of Australian Children."

2. Changing Pattern of Diseases in Children in South East Asia

   Guest Speakers:

   1. Prof. WONG HOCK BOEN (Singapore): "Concerning Important factors affecting the pattern of disease in South East Asia."

   2. Prof. SUTEJO (Indonesia): "Concerning Past, present and future pediatric problems in South East Asia."

   3. Dr. BAHRAWI W. (Indonesia): "Concerning The role of environmental factors in epidemiology of diseases in Indonesia."

   4. Dr. CHIN MAU WANG (Taiwan, Republic of China): "Concerning Changing causes of death in children in Taiwan Republic of China."

Pesticide Poisoning as a Pediatric Emergency

by

I. DARMANSIAH

Abstract

There are branches in medicine where knowledge of the average physician is deficient, such as in pollutional problems, industrial medicine and diseases caused by agricultural chemicals. Many of these involve the toxicology of substances rarely met with in medical practice.

A multidisciplinary agro-medical and industrio-medical approach is essential to understand the management including precautionary and legislative measures that have to be taken. This will involve not only medical personnel but also engineers, chemists, agronomists, ecologists and many others.

Poisoning with agricultural chemicals most often involve O.C. and O.P. compounds. In adults these cases have been typified by self-poisonings and through neglect of safety measures prescribed by the factory. In children poiso nings almost always have been associated with accidents. Here the most important factor is also negligence of parents to keep away dangerous pesticides from the reach of children.

There are quite a few potent antidotes available in pesticide-poisoning, notably atropine for O.P.'s and carbamates, vitamin K for warfarin, and dimercaprol for arsenics. There are also chemicals like paraquat and pentachlorophenol for which no antidote is available and they present great problems in treatment.

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Introduction

Poisoning has always been an emergency in children as well as in adults. In children it is more often an accidental type of poisoning. In some emergency hospitals, poisoning could constitute up to 20% of emergency cases. A survey in 11 Jakarta hospitals (Darmansjah et al., 1974) showed, however, that the ratio of poisoning-admissions was only 34 per 10,000 and that pesticides-poisoning had an incidence rate of 6%. In the Department of Child Health of the Cipto Mangunkusumo General Hospital, Jakarta, poisoning-admissions were 47 in 1971, 28 in 1972 and 18 in 1973. Of these only two were due to pesticides. There is reason to believe, however, that the real incidence of poisoning is much greater. Jo Kian Tjay et al. (1971) listed 6 pesticide poisoning cases during the years 1963 — 1968 in Medan and reported 157 cases of arsenic poisoning in children living in two orphanages at an outbreak in 1969 (Jo Kian Tjay and Djohan Aziz, 1971).

There are doubt that poisoning by pesticides could pose a problem in diagnosis and treatment, and that more pediatric cases could be anticipated when pesticides are being used more abundantly in house and garden.

Organochlorine compounds (O.C.)

These are chlor-substituted hydrocarbons and are represented as DDT, dieldrin, aldrin, BHC, thiodane, endrin, chlordane, isodrin and kethane. Endrin is by far the most toxic of the series with an L.D.50 of about 7 mg/kg. It has been the cause of death in many cases of suicidal as well as accidental poisoning. O.C. insecticides are less and less used now in Indonesia and are largely replaced by organophosphorus which in itself is equally toxic if not more. Problems of persistence are however less intense with the last mentioned.

Clinical picture

All O.C. compounds have the same action in man, primarily on the central nervous system where superspinal stimulation resulting in tremors and convulsions dominates the entire clinical picture of poisoning.

About half to three hours after ingestion or absorption through the skin, tremors are manifested which will proceed into convulsions with shorter and shorter intervals and if severe, a state of continuous convulsion will be reached. Coma often accompanies severe poisoning and fever may be present. At this stage diagnosis can only be made if there is an indication of O.C. ingestion or absorption of its only solutions through the skin.

Differential diagnosis should be made against all convulsive disorders.

alerting obstetricians and other doctors with regard to all future babies in these families, have reduced kernicterus in Singapore almost to vanishing point. The annual exchange transfusion rate of 400-500, has been reduced also by utilizing light therapy (sunlight by day and blue light by night) and estimation of unbound bilirubin in the serum.

ROLE OF AUSTRALIA (AU) ANTIGEN IN CHRONIC LIVER DISEASE IN INFANCY AND CHILDHOOD: Melihat Ertugrul, Turkish J. Pediatri. 16:8-14 14 (1974).

The Australia antigen was sought in patients during childhood who had acute viral hepatitis, chronic hepatitis and cirrhosis of the liver. The incidence of positive tests for the Au-antigen in viral hepatitis was 36.2%; in neonatal hepatitis, 33.3%; in chronic hepatitis, 60%; and in cirrhosis, 41.3% as determined by the complement-fixation technique.

Some of these positive patients were found to be Au-antigen negative by the immunodiffusion test. Different Au-antigen percentages were encountered in these diseases and statistical test results seemed to be insignificant. Au-antigen is much higher in acute viral hepatitis and in chronic liver disease than in healthy control groups and patients with other kinds of liver pathology. A much higher proportion of patients (44.8%) with cirrhosis have had a past history of hepatitis or jaundice.

It would seem that the Au-antigen carrier state plays an important role in the etiology and pathogenesis of most form of chronic liver diseases.

Our study also confirmed that complement-fixation is more sensitive than immunodiffusion for detection of the Au-antigen.
wed normal morphology and complete recovery of all enzymes measured. The mean number of hospital days was 46 ± 4.8 for group A and 34 ± 1.6 for group B (p < 0.05) suggesting that patients given enteral feedings early tended to have a more rapid return of intestinal function and of some intestinal enzymes.


Cell mediated immune response was normal in normal and malnourished children.

In children with kwashiorkor and marasmus, there was a significant decrease in the number of T cells and also a significant depression in the lymphocyte response to PHA. These results indicate impaired cell mediated immunity in PCM which may partly explain the increased susceptibility to infections observed in malnourished children. However these immune responses were normal in under-nourished children. In children who had kwashiorkor in their early childhood, though there was a significant decrease in the number of T cells, the lymphocytic response to PHA was normal.

Thus the effect of early malnutrition on lymphocyte function in later life is partly reversible.


Previously, in Singapore, kernicterus was one of the major causes of death during the early neonatal period, and one of the major causes of mental retardation if the baby survived. Erythrocytic G6PD deficiency with haemolytic triggers due to moth-balled clothes and the use of herbs accounted for nearly half of the cases. A large proportion of neonatal hyperbilirubinaemia was and is still due to culture-based use of herbs which may depress liver function-the so-called liver immaturity group. A kernicterus surveillance project was started in 1965 by be the Department of Pediatrics, University of Singapore, utilising mass screening of all newborns for G6PD deficiency, and isolation of these babies for a period of 3 weeks away from haemolytic triggers, together with doctor and public education via the mass media, and pin-pointing families at risk and

Laboratory findings

The only laboratory proof of O.C. poisoning is the demonstration of its presence in the blood. It is convenient to use a thin layer chromatography technique, which is able to detect at least 1 ppm DDT in the blood or DDA in urine.

A two and a half year old child who ingested a spoonful of DDT by mistake was admitted in the Pediatric department last year and had a blood level of 15 ppm, four hours after ingestion. Severe convulsions were noted but treatment brought complete recovery.

Treatment

There is no specific antidote for O.C. insecticide poisoning, but symptomatic treatment of convulsions and anoxia may be lifesaving.

a. General measures: All clothes soaked with the insecticide should be removed from the body and the skin washed with soap and lots of water. After oral ingestion, the insecticide should be removed from the stomach by gastric lavage or vomiting. Great care should be taken in these procedures because of the danger of aspiration, especially in children. The patient should be placed on his left side with the head in a downwards position using a large enough gastric tube. Lukewarm tap-water can be used for stomach-washing, repeating the procedure 20-30 times. In general, gastric lavage is still useful if the agent has not been more than 4 hours in the stomach.

To hasten passage through the gut, a solution of an appropriate concentration of magnesium sulfate can be left in the stomach. Oily laxatives are contraindicated since they may promote absorption of the insecticide from the gut.

b. More specific measures:

(1) Convulsions in general cause anoxia, so that oxygen is needed. No mouth to mouth breathing is allowed.

(2) Anticonvulsant therapy is essential when frank convulsions occur or for prevention when hyperexcitability exists. Pentothal sod. or diazepam i.v. can be used to control status convulsivus immediately, but care should be taken to administer only a dose sufficient to stop the convulsions. Phenobarbital can be administered to control further convulsions and has the advantage that it is an enzyme inducer and thus enables the liver to metabolize DDT at a faster rate.

(3) Morphine is contraindicated because of the danger of respiratory depression.

(4) Epinephrine or other vasoconstrictors may not be given to combat possible shock, because these can cause arrhythmia of the heart.
due to sensitization by organochlortines.

(5) All cases must be hospitalized for at least 48 hours after convulsions have ceased and phenobarbital oral treatment should be extended for a longer period. This must only be withdrawn gradually.

Organophosphorus compounds (O.P.)

Starting off as chemicals meant to kill man in war, these phosphate esters were further developed to serve mankind in the struggle against food shortages and disease. Its deadly characteristic against the human race is, however, retained.

Available in Indonesia are among others: Malathion, Dimecron, Ba-zutin, DDVP, and Diazinon. Pharmacologically they all act by the same mechanism, inhibiting cholinesterases (ChE) persistently and hereby leaving acetylcholine (Ach) unhydrolyzed. This results in accumulation of Ach at all sites where cholinergic transmission is physiological.

To summarize, the actions of Ach in the body are stimulations of:

(1) muscarin receptors (smooth muscles and exocrine glands)
(2) nicotinic receptors (autonomic ganglia and striated muscles)
(3) adrenal medulla (secretion of epinephrine)
(4) synapses in the brain.

The high fat-solubility of these O.P. compounds allows free absorption through all mucous membranes and the intact skin.

Clinical picture

A pure clinical diagnosis of O.P. poisoning can be made when we combine history with the presence of cholinergic effects. Each history of contact with O.P. compounds must lead to suspicion of poisoning when symptoms arise.

The first manifestations will be notable within a few minutes to two hours after exposure.

Signs and symptoms can be divided into central and peripheral, beginning with non-specific ones like vomiting, feeling of weakness, headache, visual disturbances and ataxia.

These quickly pass into the following important muscarinic signs: hypersecretion of saliva, sweat, nasal and bronchial mucus, colic of the gut with diarrhea, myosis and bradycardia or tachycardia.

Nicotinic signs are generally biphasic: first twitchings and convulsions of voluntary muscles occur, followed by paresis or paralysis.

Central signs include: ataxia, diminished reflexes, difficulty in speech, respiratory depression, cyanosis and coma.

The cause of death at the beginning of poisoning is airway obstruction, when no measures are taken to


The relative effectiveness of phototherapy and exchange transfusion for nonhemolytic neonatal hyperbilirubinemia was compared in two closely matched groups of infants. Although the exchange transfusion achieved an immediate reduction of bilirubin level, the "rebound" was rapid and tended to offset this reduction. The more gradual and steady effect of phototherapy resulted in a significantly lower serum bilirubin level at 1, 2, and 3 days after commencement of therapy; the rebound after phototherapy was demonstrated to be more effective than exchange transfusion in achieving prolonged reduction of bilirubin levels for nonhemolytic hyperbilirubinemia. With more efficient lamps delivering more energy in the desired spectrum, it would seem feasible to treat hyperbilirubinemia of whatever etiology with this safer and more convenient form of therapy, though sometimes only as an adjunct therapy.


Two therapeutic regimens were compared in 16 infants with protracted diarrhea and malnutrition. Eight patients were treated with total parenteral nutrition given via a central vein (group A); the remaining eight patients received a combination of dilute parenteral nutrients given in a peripheral vein plus continuous enteral feedings of an elemental diet group B.

All patients recovered although two infants in group B were switched to TPN treatment after a poor response to the elemental diet.

Intestinal biopsies were performed: (1) before treatment; (2) after 2 to 3 weeks of TPN or elemental diet; and (3) after 2 to 3 weeks of Nutramigen feedings. Before treatment, all patients had atrophic changes in the jejunal epithelium and deficient disaccharidase and trypsin activities.

The second biopsy showed morphologic recovery in all patients, incomplete recovery of lactase and trypsin in both treatment groups, and complete recovery of sucrase and maltase activities only in group B patients. The third biopsy sho-

Mortality and morbidity in gastroenteritis in children is the result of fluid loss, acid base and electrolyte imbalance. Hence therapy should be aimed at preventing or correcting this imbalance. The use of intestinal antibiotics to treat the condition should not be a routine, but parenteral antibiotic therapy in the presence of septicaemia is mandatory.


Data have been accumulated to determine the prevalence of yeast colonization of the skin and digestive tract of hospitalized infants and children. There was no difference in the prevalence between hospitalized patients at the time of admission and nonhospitalized children. However, there was a positive correlation of the duration of hospitalization and the prevalence of yeast colonization. There were no correlations of hospitalization with site of colonization, age of the patient, or type of yeast isolated.


The incidence of necrotizing enterocolitis in the newborn infant has increased within the same time period that increasing emphasis has been placed on oral alimentation of very small infants. A prospective investigation was conducted to determine the nutritional efficacy as well as the incidence of necrotizing enterocolitis of a standard cow milk formula compared with an elemental formula. Sixteen infants who weighed less than 1,200 gm were randomized and fed one of the two formulas. The clinical status of the two groups was similar. Seven of eight (87.5%) infants fed the elemental formula and two of eight (25%) fed the standard cow milk formula developed necrotizing enterocolitis (p < 0.02). The hypertonicity of the elemental diet may have contributed to the increased incidence of necrotizing enterocolitis in infants fed this formula.

 remove mucus from the upper respiratory tract. Later on central and peripheral respiratory depression or brain damage may lead to lethal complications.

Hypersecretion of exocrine glands are important indices although sweating might not be prominent in air-conditioned rooms.

The occasional presence of mydriasis sometimes misleads diagnosis. This might be due to apprehension associated with poisoning (sympathetic stimulation) or as a result of paralysis of the constrictor muscles of the iris (overstimulation).

Laboratory findings

Leucocytosis, slight proteinuria and glycosuria may be found. The ChE level of the blood is greatly reduced. Normal ChE levels are 75-100% of a man's statistical preexposure value. Liver insufficiency can cause lowering of these figures.

The reserve capacity of ChE is of such a magnitude that symptoms only occur when there is less than 30—35% in the red blood cells.

The prognosis of patients with practically zero ChE activity is by no means poor when vigorous treatment is started as soon as possible.

Treatment

(1) Artificial respiration preferably with a mechanical respirator is life saving, including clearing of the airway passage by suction. Mouth-to-mouth breathing is prohibited. A serious poisoning has occurred to a doctor who applied mouth-to-mouth breathing in a case of parathion poisoning.

(2) If O.P. has been spilled on the skin, wash immediately with soap and water.

(3) If needed and the patient is conscious, induce vomiting or gastric lavage.

(4) Administer atropin sulfate 0.04 mg/kg B.W. intravenously and repeat every 10-15 minutes until complete atropinization is accomplished, which includes flushing of the face, tachycardia, dry skin, diminished bowel-sounds, decreased secretion of saliva, and mild miosis.

When atropinization is adequate, coma and labored breathing will usually be overcome. The patient should be watched constantly, as within a few hours the signs may reappear and further atropinization may be required. There is no maximum dose for atropine, dosage should be guided by reappearance of signs and symptoms. Atropine will restore respiration, muscular and central signs; while convulsions or paralysis are not affected.

(5) Some oximes have the ability to regenerate the phosphorylated ChE. There are: 2-PAM iodide (2-pyridine-aldoxime-methiodide) or Protopam or pralidoxime iodide; dia-
cetyl monoxide (DAM) and 1,1-tri-methylene-bis-(4-formyl pyridinium bromide) dioxime (TMB-4).

Pralidoxime is the most widely used, in conjunction with atropine. The dose is 10-20 mg/kg B.W. administered slowly intravenously. This dose may be repeated several times at 20-minute intervals when paralysis is not adequately affected. There is no benefit if pralidoxime is administered after more than 36 hours because of a dealkylation process which is called "aging".

Carbamates

Carbamate insecticides (such as Carbaryl) resemble prostigmin in structure and are reversible ChE — inhibitors. Unlike prostigmin, they are very well absorbed through the intact skin, inhalation or digestive mucosa.

All the actions and toxic properties are like those of the O.P.'s, but are usually of more sudden onset and less prolonged. Because ChE-depression is not persistent there is less danger of cumulative effects.

Blood- cholinesterase determinations are of little use as the carbacholase enzyme very rapidly dissociates. Treatment consists of the measures taken for O.P.'s with the exception that 2-PAM is not required. Atropinisation is reached with a smaller total dose and relapse after atropinization occurs less frequently.

**Warfarin**

Warfarin, a hydroxycoumarin derivative is a slow-killing rodenticide used as bait. It acts by inhibition of prothrombin formation and capillary damage. The ingestion of about 1.2 mg/kg bodyweight per day for about 15 days has resulted in serious illness of 14 persons of whom 2 died.

Finished bait usually contains 0.025% active material.

**Clinical picture**

The inhibition of prothrombin only becomes apparent if the reserves in the body are consumed. Owing to this fact bleedings occur after repeated ingestions or a single massive dose, usually after a few days or weeks. When massive doses are ingested abdominal pain is evident directly.

Bleedings usually start as epistaxis, intestinal bleeding and petechial rash. These become worse and involve larger areas like in ecchymosis or hematomas and massive bleedings especially at the elbows, knees and buttocks. Hematuria may be present. Complicating phenomena are cerebral hemorrhage and hemorrhagic shock.

**Laboratory findings**

The principal detectable change is the demonstration of reduced prothrombin activity in plasma. The blood-clotting time and bleeding time

**Abstracts**


Forty-two infants with persistent diarrhoea were fed intravenously using a simplified regime based on Intralipid and an amino acid, Fructose and ethanol solution. Peripheral veins were used for up to 56 days, and with scalp veins complications were few and minor. The use of arm and leg veins caused more frequent local problems and is not advised.

Central venous lines became necessary in 5 infants, and 3 developed septicaemia. The regime was well tolerated with adequate weight gain when intake was adjusted to the infants' needs. Rates of infusion of 1 gm Intralipid/Kg hourly over 14 hours did not cause persistent lipaemia (except transiently in 2 infants) nor metabolic acidosis. Infants must be fully rehydrated with correction of acidosis and electrolyte imbalance before starting intravenous feeding, or acidosis and dehydration from osmotic diuresis may occur. Intravenous feeding should be started gradually and cautiously in severely malnourished infants, and should not be used where liver function is abnormal.


The diagnostic value of oral lactose and sucrose tolerance tests was investigated in 61 children. A total of 105 oral disaccharide tests were carried out. When the rise in blood sugar was low, the same disaccharide was, as a control measure, instilled directly into the small intestine through a tube. This was carried out in 40 cases. In 21 patients the rise in blood sugar following the two forms of administration was correlated with the disaccharidase activity in a peroral small-intestine biopsy. The incidence of false-positive oral lactose tests was between 23 and 30%, that of false-positive oral sucrose tests between 24 and 33%. A border value of 20 mg per 100 ml in the rise in blood glucose within the first hour following a direct intra-intestinal administration affords a very satisfactory distinction between patients with and without disaccharide malabsorption. Blood glucose determinations exceeding one hour were found to be without diagnostic value.
may be prolonged, but not necessarily. Also blood may be demonstrated in urine and feces.

Treatment

Vitamin K is a specific antidote and should be given 1 mg/kg B.W. three times on the first day of treatment. Afterwards smaller doses should be continued until prothrombin time becomes normal.

Shock should be treated by frequent small transfusions with fresh whole blood.

Any bruises or other trauma must of course be avoided, to minimize bleeding.

Parquat

Parquat is a weedkiller, marketed under different names, like "Gramoxone", "Graminex" and "Weedol". The fluid-concentrate contains 20% paraquat and is extremely toxic, while the 5% granular form is considerably less toxic. It has an unpleasant taste, so that if ingested accidentally it will almost immediately be rejected. In spite of this, deaths have been reported, indicating the high toxicity of the concentrated product.

Clinical picture

A burning sensation in the mouth and abdomen is immediately felt and ulceration of the mucous membranes occurs within a few days. A few days to a week after ingestion an irreversible proliferative alveolitis and bronchiolitis become evident clinically, while X-ray of the lungs reveals granular patches. Renal failure may develop during the course of the intoxication as well as hepatic parenchymal jaundice.

Treatment

So far most forms of treatment have been failures with the exception of peritoneal dialysis which has had some limited success. Early forced diuresis may be tried before renal failure ensues, but seemingly the lung-changes develop as the first passage of paraquat through the lungs. Cortisone has been shown not to be effective.

A lung transplant has been tried, but the patient died two weeks later, probably because paraquat was still circulating in the blood after the successful operation. An attempt may be made to inactivate unabsorbed paraquat by administering orally Fuller's earth as a 30% suspension or bentonite as a 6-7% suspension.

Arsenicals

There are two forms of oxides of arsenic, the trioxide and the pentoxide. Both form acids with water and produce their corresponding salts. Applications of arsenicals in agriculture are as rodenticides
(As₂O₃), and herbicides (arsenite and arsenate). The use as insectici-
dal baits are not popular.

All arsenical pesticides are general protoplasmic poisons and act by
binding sulfhydryl groups. Enzymes like pyruvate oxidase and phospha-
tases are hereby inhibited and impair tissue respiration. It is an ir-
ritant to the mucous membranes of the digestive tract regardless of its
site of absorption, whether from oral ingestion or inhalation.

Damage to the capillaries is a pronounced manifestation, causing dil-
ation and increased permeability. These in turn will cause congestion
of blood, thrombosis, ischemia and finally necrosis of tissues affected.

The fatal dose is around 1 mg/kg B.W. of arsenic trioxide; finer dis-
ersion will cause greater absorption and toxicity.

Clinical picture

Arsenic pesticides-poisonings are almost always acute and often in-
volve children. Abdominal pain, vomiting and diarrhea resembling
cholera are the most important signs. Dehydration is the result of
profuse fluid-loss.

If the patient survives, usually
after 3-14 days, exfoliative derma-
titis and polynuertis with transverse
bands on the nails are found as se-
quel. Liver and renal degeneration
may be noted, and rarely cardiac
involvement.

Laboratory findings

Although normal people have arse-
nic in their body and excrete it in
their urine (as high as 0.17 ppm as
As₂O₃), poisoning cases definitely
show a higher concentration in urine,
blood or tissue levels. Urinary arsenic
concentrations in poisoning are in the order of 4-6 ppm per day.

Liver and renal function tests may
show abnormality and the urine may
contain albumin and hemoglobin.

Treatment

As with all types of acute poison-
ing, gastric lavage is indicated when
the poison is ingested not more than
4 hours previously and the patient
is not unconscious.

Symptomatic treatment should be
mainly instituted towards dehydra-
tion with saline infusions. Dimer-
caprol (BAL) as 10% solution in oil
is a specific antidote and acts by che-
Iating As into a more soluble com-
plex, so that excretion through the
kidney is enhanced. BAL is given
intramuscularly at the following sche-

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va, 1968.
tion education, food production and distribution, infant feeding practices in various parts in Indonesia, food preservation etc. The purpose of this nutrition club is to improve cooperation and to achieve better understanding of the many factors, which play a role in malnutrition. It could also help us to keep informed of recent problems, progress and activities in the field of malnutrition. Another suggestion is intensification of nutrition education in the medical schools.

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First 2 days: 3 mg/kg B.W. every 4 hours.

Third day: 3 mg/kg B.W. every 6 hours.

Thereafter: 3 mg/kg B.W. twice daily until recovery.

Pentachlorophenol

Pentachlorophenol (PCP) is soluble in various fat solvents, but the sodium salt which is more often used, is freely soluble in water. Applications in agriculture are as weed-killer, defoliating and wood-preservative. The last mentioned is applied in Kalimantan for preservation of very inferior sorts of wood. The dip-fluid consists of:

5 kg sod. pentachlorophenate
28%

1 kg Lindane
28%

1 1/2 kg borax

In 2001 water

Of these ingredients PCP is by far the most toxic. It has a lethal dose of about 5-50 mg/kg B.W. Poisoning has occurred through absorption by the skin, inhalation or ingestion.

The main action in man is the uncoupling of oxidative phosphorylation so that the metabolic rate is increased markedly, resulting in a rise of body temperature. So far all PCP intoxications were either suicidal or occupational and for this reason involve only adults. Pediatric cases however could show up as an accident or the involvement of children in such work.

Clinical findings

The onset of critical illness is acute and the course progressive. Cummulation however can precede this sudden illness without other warning than aspecific symptoms like weakness, anorexia and headache.

Tightness in the chest, sweating and dehydration may be marked and the temperature frequently goes up very high which differentiates this condition against O.P. poisoning. Since PCP resembles phenol, acidosis is a prominent finding. Coma occurs very early in severe poisoning.

Laboratory findings

Nothing conclusive could be detected in the ordinary laboratory investigations. PCP can be detected in urine and blood and in fatal cases urine levels have ranged from 55 — 96 ppm.

Treatment

Treatment is purely symptomatic directed towards fluid and electrolyte balance and the lowering of body temperature with ice packs. Atropine is contra-indicated.


has been declared as a public health problem and the primary cause of blindness in Indonesia (Seminar on blindness, Bandung 1964).

Xerophthalmia was found to be a cause of blindness in:

31.7% of all causes of blindness in Ophthalmologic Department, Dr. Soetomo General Hospital (Tamin Radjiman and Oka, 1972).

40% in Ophthalmologic Hospital Undaan (Ten Doesschate, 1968).

17% in Ophthalmologic Hospital Undaan (Bausuki, 1973).

The significant decrease in incidence, found by Bausuki (1973), after a period of 11 years, is probably due to intensification of prophylactic administration of vitamin A and nutrition education. Addition of green leafy vegetables, which belong to the cheapest foodstuffs, to traditional infant food, like rice and banana, should be encouraged. Often eggs are sold instead of given to the infant.

2. Indirect Hazards:

These arise as a consequence of the direct hazards to the malnourished infant and affect:

a. Its family
b. The society.

Impairment of learning ability and behavior will be felt when the malnourished child has grown up. Its effect on the family and on progress and development of the country has been discussed.

Decreased resistance to infection and a predisposition to diarrhea combined with poor hygienic conditions among the low socioeconomic group will cause recurrent often chronic infections. This necessitates frequent visits to the clinic or even hospitalization in already crowded out-patient departments and pediatric wards. It also means expenses for treatment and special care, leading to neglect of other children in the family. The mortality rate is high. Among infants admitted with PCM in the Pediatric Department of the Dr. Soetomo General Hospital, 11% died.

Vitamin A deficiency is an important cause of blindness among children up to 16 years of age (Ten Doesschate, 1968; Tamin Radjiman and Oka, 1972).

Blindness will handicap the infant for the rest of his life. The burden is also felt by the family and by the society. Special schools for the blind and reha-