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spica. On examination, the child was drowsy but arousable on painful stimuli. The pulse rate was 82/min and the blood pressure 94/68 mm Hg. She had pin point pupils and excessive secretions in oral cavity. Auscultation revealed bilateral crepitations and occasional ronchi.

The clinical features were consistent with a diagnosis of organophosphorus poisoning. However, no history of drug ingestion could be obtained. A careful examination revealed greenish stains over the hip spica. On direct questioning, the parents revealed that they had been applying Tik-20 (Fenthrion) to kill the bed bugs which had infested the inner cotton lining of the plaster cast, which lead to slow and sustained absorption of the poison. The cotton removed from the cast had the characteristic odor of an organophosphorus compound.

The spica was immediately removed and the skin thoroughly washed with soap and water. Systemic toxicity was managed with Inj. Atropine and 2-PAM. There was rapid improvement in the sensorium after administering 2-PAM. There was complete recovery of all symptoms in the next 6 hours. Atropine was continued for 72 hours and gradually withdrawn over the next 48 hours.

A fresh hip spica was applied and child discharged. The hazards of the practice adopted to kill parasites, were explained to the parents.

### Discussion

As a group, the organophosphate insecticides are highly toxic chemicals that are rapidly absorbed by all routes, viz., respiratory, gastrointestinal, ocular and dermal(1). Absorption of poison through intact skin without causing local irritation has

been reported to be fatal(2). There is no report in the literature of the peculiar mode of poisoning seen in our case. A prompt and early diagnosis resulted in a favorable outcome.

This case highlights the extent of ignorance about the lethal chemicals which are commonly used by agriculturists in rural India. Masses need to be provided adequate information about these poisons, so that such accidents are avoided.

### REFERENCES

- 1. Taylor P. Toxicology. *In:* Goodman and Gilman's—The Pharmacological Basis of Therapeutics, 8th edn, Vol. II, 1989, pp 140-141.
- 2. Lester M, Janes F. Clinical Management of Poisoning and Drug Over Dosage, 2nd edn. Philadelphia, W.B. Saunders Company, 1990, p 1076.

# **Acute Mercury Vapor Poisoning**

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Campbell in 1948 reported first case of elementary mercury vapor poisoning(1). The recent high price of gold has stimulated many persons to extract gold from ore by forming a gold mercury

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Received for publication November 8, 1990; Accepted August 28, 1991 amalgum(2). Mercury vapor poisoning may occur when concentration of metallic mercury in air is 0.5 mg per cubic meter(3). We report a case of acute mercury vapor poisoning, which occurred accidently while extracting gold from gold mercury amalgum.

# Case Report

A 3-year-old boy presented with complaints of irritability, restlessness, nausea, vomiting and breathing difficulty for one day. Breathing difficulty was gradually increasing. There was no history of fever or foreign body inhalation. There was a history of exposure to mercury fumes. The father was isolating gold from dust. He took dust and mixed with 10 g mercury. Then he started heating the amalgum in an open utensil in a closed room. In the room his wife and two children were also present. All four family members suffered from toxicity. One year old female child expired with breathing difficulty and distension abdomen within 6 hours of exposure at home. Father and mother developed nausea and vomiting.

On physical examination, pulse, respiration and temperature were 160/min, 62/ min and 98.4°F, respectively. His blood pressure was 100/50 mm Hg. The child had wheezing and tachypnea without cyanosis. It was associated with indrawing of suprasternal, supraclavicular and subcostal spaces. Trachea was centrally placed. Percussion note was equal on both sides. Auscultation revealed equal air entry on both sides with few expiratory rhonchi. On 6th day, the child developed dysentery. Liver was just palpable. Spleen was not palpable. his hemoglobin was 10.6 g/dl. Total leucocyte count was 10,200/cu mm with 67% polymorphonuclear leucocytes. Urine examination revealed 7 WBC per high power

field and one plus albumin. Urine was sterile on culture. Stool examination revealed mucus and 4 WBCs per high power field. Creatinine was 1.9 mg/dl on admission and 1.03 mg/dl on 6th day of admission. X-ray chest-PA view revealed diffuse infiltration in both lung fields (Fig.). Skiagram chest repeated on 6th and 10th day showed clearance. Electrocardiogram showed sinus tachycardia. The child was managed with intravenous fluid, oxygen, prophylactic antibiotics, chelating agent and hydroxyzine. As we could get only two injections of BAL, therefore D-penicillamine was given for the next ten days.

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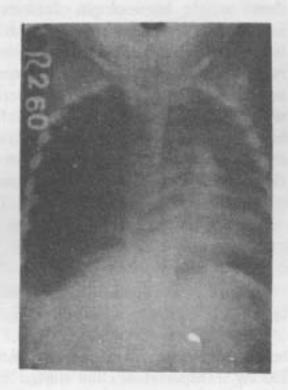
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## **Discussion**

Mercury is a liquid metal. It is volatile at room temperature. Its boiling point is 356.9°C. Metallic mercury that is not heated, usually does not cause a toxic reaction even in large amount, when given either parenterally(3) or orally(4). Mercury absorbed from respiratory tract gets oxidized in vivo to mercurous and mercuric ions' Both these ions are toxic(5). The exposure to high concentration of mercury vapor causes pulmonary irritation, nausea, vomiting, diarrhea, acute renal failure, hepatocellular dysfunction, irritability, delirium, seizures and ataxia. The mercury vapors get absorbed from distal airways completely. It causes an exudative alveolar and interstitial edema, erosion and desquamation of epithelium. The ensuing obstruction is proportionately greater in infants than in adults. It results in alveolar dilatation, interstitial emphysema, pneumatocele, pneumothorax and medistinal emphysema. Death among severely affected infants has been a rule(1,6). The literature contains 11 case reports, describing such poisoning in children(7).

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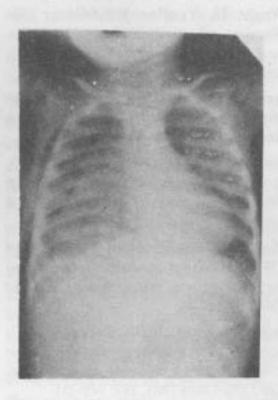


Fig. Both lung fields showing diffuse infiltration.

Management includes intensive care support(8) and administration of intravenous fluids, oxygen, prophylactic antibiotics and chelating agents. British antilewisite (BAL or Dimercaprol) is used as a chelating agent. It is an oily preparation and available in concentration of 50 mg/ml. It is given in a dose of 5 mg/kg for first injection and 3 mg/kg every four hourly for two days; this dose is then tapered to every six hourly for one day and then every 12 hourly for seven days. It protects kidneys if given within three hours of ingestion. If BAL is not available D-penicillamine can be used for treatment in dose of 100 mg/kg in four divided doses to a maximum of 1000 mg per day, though it is less effective(9). Hydroxyzine and chlorpromazine may be used for restlessness and tolazoline may be given for tachycardia.

Mercury poisoning is a professional hazard for goldsmiths and those dealing with gold and silver ornaments. We feel that community at risk should be taught the right procedure for purifying or extracting precious metal. Most important is that the room should be well ventilated when mercury is used for extracting these metals.

### REFERENCES

- Campbell JS. Acute mercury poisoning by inhalation of metallic vapor in an infant. Can Med Assoc J 1948, 58: 72-75.
- Sndograss W, Sullivan JD, Rumuk BM, Hushimoto C. Mercury poisoning from home gold ore processing. JAMA 1981, 246: 1929-1931.
- Modi NJ. Mercury. In: Modi's Textbook of Medical Jurisprudence and Toxico-

- logy. 20th edn. Bombay, N.M. Tripathi Private Ltd, 1977, pp 549-558.
- 4. Wright H, Yoeman WB, Carter GF. Massive oral ingestion of elemental mercury without poisoning. Lancet 1980, 1: 206.
- 5. Iyer K, Goodgold J, Eberstein A, et al. Mercury poisoning in a dentist. Arch Neurol 1976, 28: 788-790.
- 6. Matthes FT, Krischner R, Yow MD, et al. Acute poisoning associated with inhalation of mercury vapor. Report of four cases. Pediatrics 1958, 22: 675-688.
- 7. Moutinho ME, Tompkins AL, Rawland TW, et al. Acute mercury vapor poisoning: Fatality in an infant. Am J Dis Child 1981, 135: 42-44.
- 8. Jaffe KM, Shurtleff DB, Robertson WD. Survival after acute mercury vapor poisoning. Role of intensive support care. Am J Dis Child 1983, 137: 749-751.
- 9. Akabane T. Acute and chronic mercury poisoning. In: Nelson's Textbook of Pediatrics, 13th edn. Eds. Behrman RE, Vaughan VC, Nelson WE. Philadelphia, WB Saunders Co, 1987, pp 1505-1507.

# **Acute Carbamazepine Poisoning**

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Carbamazepine is an anticonvulsant useful in the treatment of tonic clonic sei-

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Received for publication February 12, 1991; Accepted August 28, 1991 zures and partial seizures with complex symptoms. Patients on long term therapy show mainly hematologic, dermatologic and hepatic derangements. There are very few cases reported, of acute toxicity mainly in adults, showing chiefly cardiovascular and neurologic dysfunctions(1-4). Here, we report our observations on a child with carbamezepine poisoning with a brief review of literature in relation to clinical spectrum and management of acute carbamezepine intoxication.

## Case Report

A 3-year-old female child weighing 11 kg was admitted with a history of ingestion of eight tablets (200 mg each) of carbamezepine prescribed for her mother. During transportation child started having tonic clonic seizures. On examination she was mainly responsive to deep painful stimuli and was having intermittent decerebrate posturing. Vital signs were maintained. Pupils were semidilated and sluggishly reacting. Meningeal signs were absent. Doll's eye response was absent. The gag reflex was diminished and tendon reflexes were not elicitable. Plantar response was flexor bilaterally. Chest examination revealed evidence of bronchopneumonia in the right inframammary region. Other systems were essentially normal.

Laboratory investigations revealed hemoglobin of 8.3 g/dl, TLC of 7000/cu mm, DLC P<sub>60</sub> L<sub>38</sub> E<sub>1</sub> M<sub>1</sub> and platelets were reduced to 60,000/cu mm. Lumbar tap was normal. Blood culture was sterile. X-ray chest revealed right lower zone pneumonia probably secondary to aspiration of vomits enroute to hospital.

Stemach wash was done. Patient was treated with antibiotics, and intravenous fluids. Vitals were monitored. Convulsions