Letters to the Editor

Aluminium Phosphide Poisoning: A Growing Concern in Pediatric Population

Aluminium phosphide (ALP), a cheap and freely available pesticide, has emerged a common agent responsible for poisoning in children with a resultant high mortality(1). The poisoning is usually accidental in children but may be suicidal in adolescents(2).

Twenty cases of ALP poisoning aged between 7-12 years who were admitted in the Pediatric Emergency Services between July 1995 to July 1996 were evaluated. The diagnosis was made on the basis of the history of ingestion and positive silver nitrate tests on gastric lavage fluid. All routine investigations including, serum electrolytes, liver function tests, renal function tests, acid blood gas analysis and close electrocardio-graphic monitoring, were done in these cases. Gastric lavage, activated charcoal, antacids, intravenous ranitidine and management of shock (intravenous fluid + dopamine + hydrocortisone) and relevant therapy for cardiac arrhythmias, anemia, seizures and metabolic acidosis were the mainstay of treatment. We observed hypo-magnesemia in all the 20 cases and magnesium sulphate in a dose of 200 mg/kg intravenously (6 hourly or 4 hourly, depending upon the severity) was used. Out of 20 cases, 12 (60%) had presented with shock, 4 (20%) with hematemesis and malena, 2(10%) with hematemesis and shock and 2 (10%) with acute respiratory distress (ARD). The overall mortality was 55%; the rate rose to 100% in patients with ARD, and 50% in each group of shock, hematemesis with shock, and hematemesis with malena. The mortality had a direct relationship with the time of presentation to the hospital after ingestion of the poison; mortalities were 14% of 40% and 89% (8,9) for time gaps of presentations of below 4 hours, 4-8 hours and above 8 hours, respectively.

ALP on exposure of moisture and hydrochloric acid of stomach liberates phosphine gas which gets absorbed quickly by simple diffusion and causes tissue hypoxia due to non-competitive inhibition of cytochrome oxidase system of mitochondria or damage by free radicals(3). Global hypoxic myocardial injury and peripheral capillary leakage, local irritant effects of phosphine on gastrointestinal tract and direct alveolar damage results in shock, hematemesis and malena and ARDS, respectively.

The mortality in ALP poisoning is reported to vary from 37% to 100% and depends on a number of factors like delayed arrival in hospital, duration, shock, anemia, arrhythmias, dose of poison and freshness of tablets(l). Magnesium sulphate has been reported to be effective in reducing mortality(4) but some workers(5) had found no significant differences in dose related mortality rates in patients treated with and without magnesium sulphate.

In our study, survival was better in patients who presented within 4 hours and intravenous Magnesium sulphate was started. Considering the high mortality and lack of specific antidotes, we feel that magnesium sulphate therapy should be instituted early in all patients with ALP

poisoning based on serum magnesium level.

Utpal Kant Singh,
Bhaswati Chakraborty,
Rajniti Prasad,
Upgraded Department of Pediatrics,
Patna Medical College,
Patna 800 004.

REFERENCES

1. Chugh SN, Arora BB, Malhotra GC. Incidene and outcome of aluminium phosphide poisoning in a hospital study. IndianJ Med Res 1991; 94: 232-235.
2. Sharma A, Gathwala G. Oral aluminium phosphide poisoning in Indian children. J Trop Med Hyg 1992; 92:221-222.
3. Chefurka W, Kashi KP, Bond EJ. The effect of phosphine on electron trans port of mitrochondria. Physiol 1976; 6: 65-84.
4. Avasthi R, Sharma R. Aluminium phosphide poisoning and magnesium sulphate therapy. J Asso Phy India 1994; 42: 670.
5. Siwach SB, Singh P, Ahlawat S, Dua A, Sharma D. Serum and tissue magnesium content in patients of aluminium phosphide poisoning and critical evaluation of high dose magnesium sulphate therapy in reducing mortality. J Assoc Phy India 1994; 42:107-110.

Neonatal Skin Lesions

A prospective study was conducted on 1046 consecutive babies delivered in a referral teaching hospital to determine the pattern of neonatal skin lesions. The babies were categorized according to the gestational age, birth weight and weight for gestational age. All newborns were examined at birth for skin lesions and were followed up every day till discharge and later on in our follow-up clinics every 15 days up to 2 months. Appropriate investigations like scraping for cytology, vesicular fluid smear, swabs for bacterial and fungal culture and skin biopsy were performed. A standard clinical format was designed to record the observations. A high incidence of transient neonatal skin lesions was observed. Erythema toxicum neonatorum (ETN) was seen in 50% of full term and 46.2% of preterm babies *(Table 1).* The incidence quoted in other studies ranges from 4.5% to 48.5%(1). These lesions developed by 24 hours of age in 13% and by 48 hours in 60% of cases. The main types of lesions seen in ETN were erythema, papules and pustules. The lesions disappeared within 72 hours in 90% of cases. Smears prepared from the vesicular fluid revealed eosinophils in all cases. Majority (89%) of babies had Mongolian spots. This incidence is higher than that reported in other studies(2). Only 3.4% of babies had diaper dermatitis. This incidence is low in contrast to the western studies(3). This is possibly due to less use of occlusive diapers by Indian mothers. Seborrhoeic dermatitis was documented in 6.8% babies. Forehead, eyebrows and scalp were involved in 90% of cases. The mean age of onset was 4 weeks. This is comparable to other studies(4). Skin lesions like erythema, induration, hemorrhage, necrosis and ulceration were detected in 6.1% of full term and 28.3% of preterm babies with the diagnosis of sepsis. Blood cultures of all the babies with such