Acute Tetraethyllead Poisoning

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Received September 16, 1968

Summary. Four cases of accidental poisoning with tetraethyllead are described. Three out of four of the patients died.

In the first case, pure ethyl fluid was accidentally ingested. Dominating the clinical picture of this patient were signs of greatly elevated intracranial pressure. Three other persons were poisoned as a group. They unknowingly inhaled tetraethyllead contained in a paint solvent they used. In these three cases, the intoxication manifested itself predominantly as a mental disorder suggestive of schizophrenia.

Gross and microscopic changes observed in the fatal cases gave evidence of a capillary vascular lesion, particularly involving the vessels of the CNS. Liver damage and less severe damage to the heart muscle and kidney parenchyma were also noted.

The distribution as well as the extent of the above mentioned lesions correlate approximately with the distribution and concentration of triethyllead in the various internal organs.

Key-Words: Tetraethyllead — Mental Disorder — Damage to Parenchymatous Organs.


Im ersten Fall trat die tödliche Vergiftung infolge irrtümlich getrunkenem Ethylfluid auf. Als klinisches Symptom entstand erhöhter intrakranieller Druck.

In den drei nächsten Fällen besaß die Vergiftung einen kollektiven Charakter und war durch den Respirationsverlauf verursacht. Zwei von den Vergifteten sind gestorben. Die Vergiftung trat infolge der Einatmung des Ethylfluids, das als Farblösungsmittel benutzt worden war, auf. Im klinischen Bild dominierten die Gehirnveränderungen.

In allen tödlichen Fällen dieser Bleitetraäthylvergiftung wiesen die Obduktionsbefunde eine Schädigung der Capillaren des zentralen Nervensystems und eine Leberentartung auf. Es wurden auch Herzmuskel- und Nierenschäden festgestellt.

Die Lokalisation und die Intensität der histologischen Veränderungen der inneren Organe stimmen im wesentlichen mit der in diesen Organen festgestellten Bleitetraäthylkonzentration überein.

Schlüsselwörter: Bleitetraäthyl — Gehirnstörungen — Schäden der Parenchymorgane.

Tetraethyllead (TEL) poisonings occur most often during the production of the substance, by its addition to gasoline, and by careless use of
it as a solvent. In comparison, poisonings resulting from intentional or unintentional ingestion of TEL are truly rare.

The clinical course of four cases of poisoning with tetraethyllead are presented. Three of the patients died as a result of the accident. The morphological and histochemical findings of these cases are also presented.

Clinical Observations

Poisoning by Ingestion

Case I. T. K., a 32 year old male with a defective sense of smell (caused presumably by an injury to the skull suffered several years before), swallowed approx. 3 spoonfuls of ethyl fluid by mistake. Immediately afterwards he drank a large quantity of water and vomited profusely. Two hours later he was admitted to the Clinical Department for Occupational Diseases.

At the time of admission the patient was conscious. He complained of headache, drowsiness and epigastric pain, and continued to vomit a reddish colored liquid. The conjunctivae of both eyes were congested. The oral cavity emitted an aromatic odor. The patient was apathetic but showed signs of motor excitement when vomiting. His blood pressure was 90/60 mm Hg; pulse was 82/min, respirations 18/min, and temperature 36.5 °C.

In spite of immediate administration of 1.0 gm of EDTA and stomach lavage with Antidotum Metallorum "Sauter", as well as intensive symptomatic treatment, the patient's condition underwent a gradual but continuous deterioration. The pupils became dilated and lost power of accommodation, the retinal vessels narrowed, tendon reflexes increased, and incontinence of excrements ensued.

Laboratory findings revealed a reduced plasma alkali reserve of 11 mEq/l and a prothrombin time (Quick) of 42 sec (control was 15 sec). Urine: Protein 0.528/g, sugar 1.9 %, and cylinders and erythrocytes were found in the sediment. Peripheral blood count: Hct. 60%, Hb 12.2 gm-%, RBC 5,200,000, color index 1.16, and WBC 10,400.

By the eighth hour after ingestion of the poison, the patient was approaching a state of coma. On the second day after admission, several episodes of beginning pulmonary edema were recorded. The pulse rate was 116—142/min, blood pressure 85/50—140/90 mm Hg, respirations 26 to 38/min, temperature 36.8—37.6 °C. Meningeal irritation signs appeared and tendon reflexes became more accentuated. Examination of the muscles showed an elevated tone and presence of fasciculations. Muscle excitability increased to actual twitching in response to moderate touch stimuli.

1 The results of chemical studies carried out in these cases were reported by Bolanowska et al.: Arch. Toxikol. 22, 287 (1967).