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Case Report Open Access

Polyvinyl Chloride (PVC) Acting as Occupational Hazard in a Factory Worker Presented with Acute Toxic Encephalopathy: A Case Report

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Abstract

Neurotoxic exposures are common and ubiquitous in the environment, particularly in occupational settings. When a patient presents with toxic encephalopathy arriving at the correct diagnosis is often a diagnostic challenge. Recognition of toxic encephalopathy is important because the correct diagnosis of occupational hazards can prevent others (e.g, workers at the same workplace) from further exposure to the toxin. Polyvinyl chloride (PVC) plastics are extensively used for a very wide range of purposes. Before PVC can be made into finished products, it always requires conversion into a compound by the incorporation of additives such as heat stabilizers, UV stabilizers, plasticizers, processing aids, impact modifiers, thermal modifiers, fillers, flame retardants, biocides, blowing agents and smoke suppressors, and, optionally pigments. Lead, cadmium or organotins are used as stabilizers. Zinc, tin stabilizers and pigments are also used in PVC. Antimony-zinc complexes have been used as flame retardants in PVC products. We here present a case with acute encephalopathy due to accidental inhalation of PVC and heavy metal combination during the manufacture of electric wire.

Keywords: Polyvinyl chloride; Encephalopathy; Antimony-zinc complexes

Introduction

The term "toxic encephalopathy" is used to indicate brain dysfunction caused by toxic exposure. It includes a spectrum of symptoms ranging from subclinical deficits to overt clinical disorders. Chemicals causing damage to the central nervous system (CNS) are ubiquitous in the environment, particularly in occupational settings. Industrial processes are major sources of some of the most well-known neurotoxins [1].

PVC was accidentally discovered first in 1835 by Henri and in 1872 by Eugen. Polyvinyl chloride (PVC) plastics are extensively use for in a very wide range of purposes, such as food wrappers, interior surfaces and covering of crops in agriculture and as the insulation on electrical cables. Before PVC can be made into finished products, it always requires conversion into a compound by the incorporation of additives such as heat stabilizers, UV stabilizers, plasticizers, processing aids, impact modifiers, thermal modifiers, fillers, flame retardants, biocides, blowing agents and smoke suppressors, and, optionally pigments. Lead, cadmium or organotins are used as stabilizers [2]. Zinc, tin stabilizers and pigments are also used in PVC. Antimony-zinc complexes have been used as flame retardants in PVC products [3]. Accidental exposure to PVC has been associated with interstitial lung disease. The central neurological effects of a combination of PVC and other plasticizers are not known. We here present a case with acute encephalopathy due to accidental inhalation of PVC and heavy metal combination during the manufacture of electric wire.

Case Report

A 28 y old male non-smoker, non-alcoholic, vegetarian by diet, worker in plastic factory from last 2 years presented to the IHBAS emergency with complaints of acute onset of irrelevant talking, agitation, restlessness, sleeplessness, slurred speech, imbalance while walking and sitting from last 3 d. Fluctuation in level of the consciousness, visual hallucinations, spasmodic abdominal pain and constipation were present from last 2 d. He had no history of fever, headache, nausea, vomiting, seizures or myoclonic jerks, weakness of the limbs/sensory loss, joint pain, oral ulcer, photosensitive rash, change of color of skin and recurrent pain abdomen. No history suggestive of cranial nerve involvement. No past history of similar illness and no history of any medical illness i.e. (renal impairment, hepatic impairment) diabetes, hypertension and thyroid illness were present.

On examination, he was conscious but disoriented to time, place, person, afebrile (98.6°F). His heart rate was 86/min. BP was 110/86 mmHg, respiratory rate 20/min, and oxygen saturation was 98% (while breathing room air). His pupils were bilateral 2 mm and were reacting to light. Cranial nerves were normal, tone in both upper and lower limb was normal, power was grossly 5/5, deep tendon jerk were brisk in both upper and lower limbs, planter bilateral extensor and he was not cooperative for sensory examination. Bilaterally symmetrical cerebellar signs were present. Rest of the systemic examination was unremarkable. As per history and examination we kept possibility of acute encephalopathy and managed conservatively.

Investigations at presentation and during hospitalization

HB-13.3 mg%, TLC9440, P86L10E1M l, platelets-1.8 Lakhs, ESR-20 mm/h, MCV-86.5 fl, MCH-30.9 pg, MCHC-35.7 g/dl, S. bilirubin-0.63 mg/dl, SGPT-84 U/L, SGOT-35 U/L, ALP-93 U/L, GGT-22 U/L,

urea-25 mg/d, creatinine-1.0 mg/dl, sodium-141 mmol/L, potassium-2.7 mmol/L, calcium 9 mg/dl, phosphorus-2.6 mg/dl, magnesium-2.6 mmol/L, FBS-88 mg/dl, PPBS-99 mg/dl, HbA lc-5.2%, ammonia-56 µg/dl (30-86), lactate-1.8 mmol/l, CSF-sugar-97 (RBS-129 mg/dl), protein-28 mg/dl, cell count- 1 lymphocyte/mm^3, staining negative, HSV-PCR-negative. ECG had normal sinus rhythm, normal axis, no ST/T changes. Chest X-ray PA view, USG abdomen with pelvis-normal. Thyroid profile, lipid profile was normal. Vitamin B 12-240 pg/ml, folic acid-13.6 ng/ml, HbsAg, HIV, Anti HCV was negative. 24 h urinary protein 96 (<150 mg/d), urine for prophobilinogen (PBG)-negative, NCV of all four limb, and bilateral PlOO latency in VEP was normal.

EEG showed generalized background slowing and MRI Brain showed (Figure 1) diffusion restriction in bilateral parietooccipital and, frontal periventricular white matter region with corresponding ADC change. No abnormal parenchymal and leptomeningeal enhancement was seen.

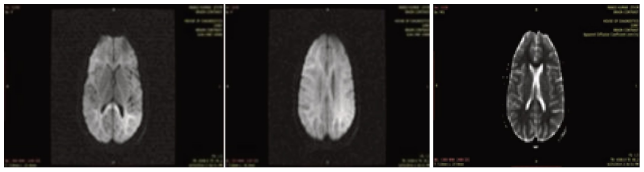

Figure 1: MRI brain showed restricted diffusion in B/L parietooccipital and frontal periventricular white matter region with corresponding ADC change. No abnormal parenchymal and leptomeningeal enhancement seen.

Blood investigations on day 3rd were unremarkable except mild rise in SGPT and hypokalemia (10^3 IU and 2.9 mmol/L), With the normal blood picture, normal CSF examination and MRI brain suggestive of demyelination we treated him I.V. Methyl prednisolone and potassium both I.V and orally.

On 3rd d his wife gave history that his colleague in PVC plastic factory also got admitted at GTB hospital with similar history last night. So we suspected possibility of acute TOXIC encephalopathy and serum sample for zinc, lead, cadmium, and mercury send. At next day his heavy metal level report showed lead (PB)-50 (N<5 µg/dl), arsenic (AS)-224 (N<15 µg/L), cadmium (Cd)-35 (<5 µg/L), mercury (Hg) below detectable level. Urine tin test was not conducted due to unavailability.

His confusional and amnesic state improved over a period of week and gradually thereafter but he remained irritable, aggressive up to 25 d. Constipation and pain abdomen was present for 1 month. Hypokalemia required I.V potassium for 2 w and oral replacement for 1 month. Marginally deranged SGPT for l month. At time of discharge from hospital he had dysarthria, truncal ataxia and delayed response to verbal commands but he was independent of his activities of daily living (ADLs). Now he is in regular follow up and last follow-up was 5 d back, in which he had gradually improving dysarthria and ataxia. MMSE was 30/30, NCV of all four limbs to see delayed neuropathy was normal. His MRI brain (Figure 2) was normal.

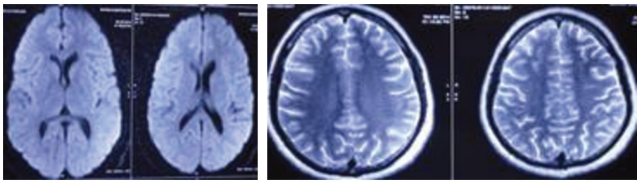

Figure 2: MRI brain.

Discussion

We diagnosed present case as acute toxic encephalopathy due to following reasons.

1) Clinical features were compatible with toxic encephalopathy and hypokalemia [l,4-13].

2) Patient was exposed to PVC plastic and corresponding heavy metals were detected in his blood.

3) Other causes of acute encephalopathy were ruled out by clinical features, blood investigations, MRI brain and CSF examination.

4) Symmetrical high signal white matter lesion was seen in brain MRI which supported toxic encephalopathy.

Acute toxic encephalopathy is a global cerebral dysfunction of rapid onset (typically days or weeks), and may be associated with alterations in the level of consciousness. The neurotoxins that cause acute encephalopathy interfere with basic cell functions in the brain [2] the causative agents include organic solvents, which can alter cellular membrane function, and gases (e.g, gas anesthetics, carbon monoxide, hydrogen sulfide, and cyanide), which can diffusely affect brain function. Heavy metals can also cause acute encephalopathies; this is more commonly associated with organic metals (e.g, methyl mercury, tetraethyl lead and organic tin) than with inorganic metals (e.g, mercury, lead and tin) [2].

The cerebral cortex is more sensitive to these toxins than is the brainstem: even when consciousness is lost, brainstem function typically remains intact.

Our patient's symptoms were due to encephalopathy and hypokalaemia. Toxicity with chemical materials such as toluene and barium had been reported as other causes of hypokalemia [5-9]. Hypokalemia can cause central pontine and extrapontine myelinolysis. Central pontine and extrapontine myelinolysis are demyelinating disorders are associated with rapid correction or overcorrection of hyponatremia. Few cases of central pontine myelinosis have been reported to have hypokalemia without hyponatremia [10,11]. Brain MRI findings of toxic encephalopathy due to various substances have been extensively described. Most of these intoxications occur through parenteral and oral intake. Inhalational poisoning resulting in CNS damage is relatively rare, with carbon monoxide being the most commonly encountered toxic agent [12] other causes are solvents and chemical materials used in the industry [14].

Polyvinyl chloride (PVC) is a synthetic polymer material (or resin), which is built up by the repetitive addition of the monomer vinyl chloride (VCM) [2,3]. Pure PVC is a rigid material, which is mechanically tough, fairly good weather, water and chemicals resistant, electrically insulating, but relatively unstable to heat and light. This can

be avoided with the addition of stabilizers. Stabilizers are often composed of salts of metals like lead, barium and calcium or cadmium, or organotin compounds [2,3]. The mechanisms associated with heavy metal toxicity have been attributed to generation of reactive oxygen and nitrogen species, which develops imbalance between the prooxidant elements and the antioxidants (reducing elements) in the body. The oxidative stress mediated toxicity of heavy metals involves damage primarily to liver (hepatotoxicity), central nervous system (neurotoxicity), DNA (genotoxicity), and kidney (nephrotoxicity) in animals and humans [15].

The pathogenesis of organotin intoxication varies. Trialkyltin targets the central nervous system and often causes demyelination. It can also cause acute toxic encephalopathy with delayed toxic effects including expansion and hyperemia of blood vessels in alba, interstitial brain edema, neurotransmitter change, inhibition of dopamine and muscarinic receptor binding to restrain adenosine triphosphatase activity [16].

Hypokalemia in our patients may be due to a number of causes. Firstly organotin impairs the function of the digestive tract, interferes the assimilation and the absorption of potassium. Secondly, poisoning of the central nervous system, especially hypothalamus, causes hypothalamic-pituitary-adrenal axis disorder and excessive release of adrenocorticotrophic hormone, which stimulates the secretion of adrenocorticotrophic hormone from the adrenal cortex. Adrenocorticotrophic hormone leads to excessive discharge of potassium by the kidney, and causes low blood potassium.

Thirdly, animal tests have shown that organotin impairs uriniferous tubule function and increases potassium discharge, leading to a low level of blood potassium [17].

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