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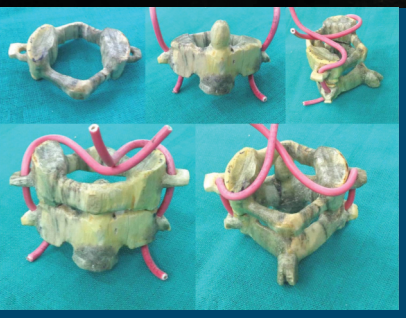

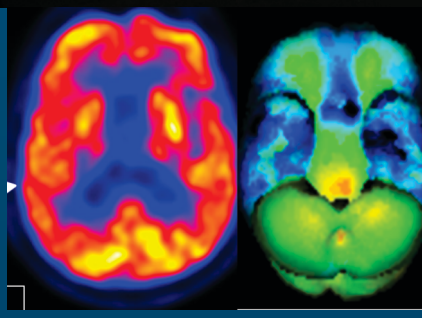

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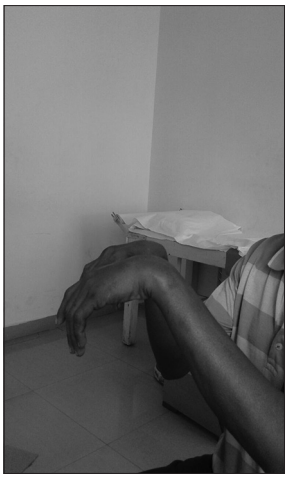

Figure 1: Patient had bilateral wrist drop

Cardiovascular Sytem	Increase in systolic and diastolic blood pressure
Renal System	Nephrotoxic- directed against PCT cells initially followed by interstitial fibrosis and seconday hypertension
Reproductive System	Infertility in men and sperm abnormalities
Endocrine System	Lowering of serum thyroxine with inappropriate rise of thyroid stimulating hormone. Delayed puberty and thelarche may be found in girls
Musculoskeletal System	Arthralgia and gout (related to nephropathy)

Non Neurological Manifestations of Lead Poisoning

Figure 2: Non-neurological manifestations of lead poisoning. PCT: Proximal convoluted tubule

A forgotten cause of bilateral wrist drop

Sir,

A 49-year-old, nondiabetic and normotensive gentleman presented to the hospital with progressive distal weakness of both the upper limbs. This started in the right upper limb for over a year at the time of presentation to the hospital and extended to the left hand after approximately 2–3 months [Figure 1].

The patient had a past history of abdominal colic approximately 2 years prior to the present illness, for which he underwent an exploratory laparotomy. However, no pathology could be found.

Lead (Pb) has a wide range of use in the industry because of its properties such as ease of casting and fabrication, resistance to corrosion, opacity to X-rays, and a low melting point.[1,2] It is gradually being replaced by other materials.

Pb is absorbed in the body from any route, that is, respiratory tract and gastrointestinal tract (elemental and inorganic lead) and even skin (organic lead). It is distributed all over the body, especially in erythrocytes. A prolonged elimination half-life (30 days in the blood and 27 years in the bone) results in an increase in its concentration in the body over time. The distribution is present in 3 pools—blood and soft tissue (exchangeable pool and thus most important toxicologically), soft tissues, and the skeletal system. It is excreted in the urine and feces (unabsorbed) as well as from the bile. Small amounts of Pb are also eliminated in other fluids e.g., saliva, sweat, breast milk, etc.[2]

Robert Kehoe suggested that the blood Pb concentrations of less than 80 μg/dl may not cause clinical Pb poisoning. Maximum blood concentration considered safe was, therefore, reduced to 80 μg/dl. At present, the safe levels are considered to be <10 μg/dl. Nephrotoxicity (seen as proteinuria and low glomerular filtration rate) occurs with blood Pb concentrations of >50 μg/dl. Sperm abnormalities occur at concentrations >40 μg/dl. Endocrinal effects are evident at concentrations >60 μg/dl.[2] Delayed puberty occurs in girls.[3]

The International Agency for Research on Cancer determined that inorganic Pb compounds are carcinogenic to humans (group 2A) and that organic Pb compounds are not classifiable as carcinogenic to humans (group 3).[4]

Pb accumulates till a critical body burden is reached, following which, sudden onset, rapidly progressive symptoms develop. However, subclinical presentations may often occur early [Figure 2].

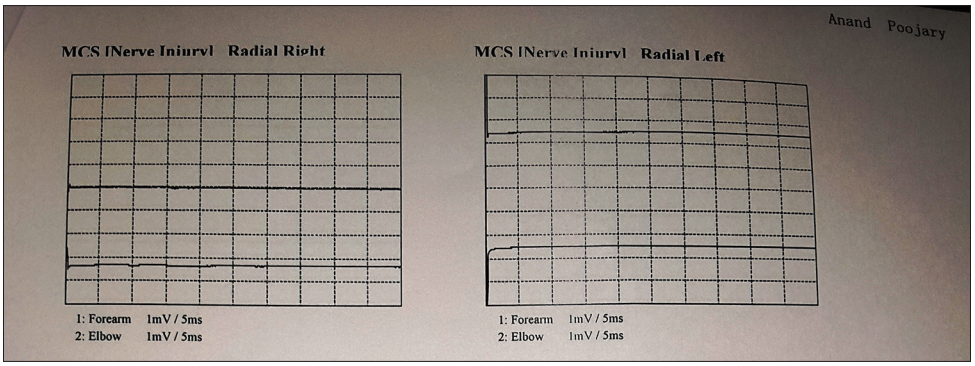

Figure 3: Electrophysiological Images showed no CMAPs

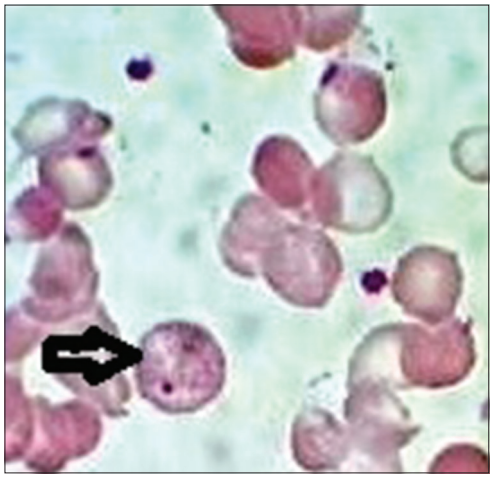

Figure 4: Basophilic stippling seen on peripheral smear

Pb may affect the central, peripheral, and autonomic nervous systems. Mild symptoms are characterized by fatigue and lethargy that can disturb the routine activities. Severe conditions such as encephalopathy, impaired consciousness, and bizarre neurological signs are unusual in adults but are frequent in children due to the occurrence of pica.[5] The severity of symptoms range from confusion and disorientation to repeated resistant seizures, coma, and death. Lateralizing signs such as focal seizures, hemiparesis, and Babinski sign on one side may also be seen.[6] Encephalopathy with headaches, excess salivation, vomiting, irritability, insomnia, delusions, and hallucinations may be seen.[7] Chronic exposure to Pb causes psychiatric symptoms and mild cognitive impairment.[5]

Neuropathy is usually asymmetrical and predominantly motor, but rarely sensory. The weakness is more common in the upper limbs than in the lower limbs, affecting finger extensors followed by wrist extensors leading to "wrist drop." Weakness may also be seen in other muscles and even in more proximal muscles. Similar weakness in lower limbs, causing a "foot drop," may be seen in children.[5]

Our patient was a factory worker in a battery manufacturing unit and had been exposed to Pb plates. The patient had held the job for the past 6–7 years. Nerve conduction velocity showed changes in the bilateral radial and medial nerves with involvement of both the lower limbs [Figure 3]. Laboratory investigations showed basophilic granules suggestive of Pb poisoning [Figure 4]. Biochemical measurements for Pb estimation were available, and the levels were found to be high (104.7 pg/dl; acceptable range upto 10 pg/l). Pb poisoning is not a common condition any more. The usual presentation is a unilateral wrist drop. Our patient showed bilateral wrist drop. Sensory involvement, usually rare, was also seen in both the lower limbs. Pb should, therefore, be considered as an implicating condition in a patient presenting with bilateral wrist drop.

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Conflicts of interest

There are no conflicts of interest.

Saumya H. Mittal, Shivanand Pai, K. C. Rakshith, Z. K. Misri

Department of Neurology, Kasturba Medical College Hospital, Mangalore, Karnataka, India

E-mail: saumyamittal1@gmail.com

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