An update on lead-related nephrotoxicity

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Nephropathy is often a fatal progressive disease with lack of appropriate managements such as dialysis or medicaments (1). Lead intoxication has been recognized as a public health risk, mostly in developing countries. It is known as a potent occupational toxin through various sources such as gasoline, paints, containing pipes, industrial processes like lead smelting and coal combustion, battery recycling, grids and bearings (2). Lead exposure can result from ingestion, inhalation and direct skin contact with compounds lead or reabsorption from a residue bullet in joints that often presents gradually. Nowadays, unusual causes of chronic lead poisoning have been reported such as adulterated opium and marijuana (2,3). Lead is a type of metal that like most toxic heavy metals, interferes with many body processes and involves many organs and tissues. Its toxicities makes various deleterious effects on the peripheral, central nervous system, hematopoietic and kidney (4). If lead poisoning does not treat on time, patients are characterized by encephalopathy with the major symptoms like irritability, poor attention, headache, tremor, loss of memory, hallucinations, delirium, lack of coordination, ataxia, paralysis, convulsions and coma (2). Lead can involve the hematopoietic system through restraining hemoglobin synthesis. It also reduces the life span of circulating erythrocytes by increasing the fragility of cell membranes acute high level lead poisoning leading to hemolytic anemia while frank anemia is associated with the blood lead level when elevated for prolonged periods (2,4). Around late 19th century, physicians knew that lead poisoning caused chronic renal failure (5). This opinion was supported by several recent studies too (1-3). According an autopsy study, the content of lead in skull bone was higher in patients who died from end-stage renal disease of unknown causes, compared to those who died of end-stage renal disease from known causes (6). On the other hand, the higher mortality rate from lead workers from end-stage renal disease, particularly in workers who were very highly exposed was detected (7). Furthermore, there was reported that, long-term lead exposure resulted to accumulation of iron in the kidney (8). The blood lead level is easy to measure. There are effective chelators like 2,3-dimercaptopropanol, meso-2,3-dimercaptosuccinic acid and calcium disodium edetate are available for mobilize deposits of lead into the urine (9). Unfortunately, exposure to lead is often considered when the patient's history precisely pointed to usual sources of lead, so screening for this disease remains silent. Additionally, there are many challenging diagnosis compete with this disease which combined with the complexity of the presentations. The data from Iran show that the most of people were in great danger of exposure and lead toxicity (3). Thus, it is a purpose that lead screening test be considered for every patient with sub-acute and chronic renal failure or nephropathy with unknown etiology, particularly beside other signs and symptoms of lead poisoning such as abdominal pain, constipation and anemia. In this regard, designing programs are necessity for management and treatment of patients.

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