Intracranial Calcification in Adults with Chronic Lead Exposure

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*AJNR Am J Neuroradiol* 1985, 6 (6) 905-908

http://www.ajnr.org/content/6/6/905

This information is current as of October 18, 2023.
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Computed tomographic (CT) findings of cerebral and cerebellar calcification are described in three American adults with raised serum lead levels and known exposure to lead for 30 or more years. Calcification patterns were punctiform, curvilinear, speck-like, and diffuse and were found in the subcortical area, basal ganglia, vermis, and cerebellum. Admission serum lead levels ranged from 54 to 72 µg/dl (normal, 0–30 µg/dl). Nonspecific neurologic manifestations consisted of dementia, diminished visual acuity, peripheral neuropathy, syncope, dizziness, nystagmus, easy fatigue, and back pain. Two patients developed chronic renal disease and hypertension; in both cases, serum parathormone was elevated. Blood, calcium, and phosphorus were normal in all three. No other structural abnormalities were observed with CT. Although the pathophysiologic mechanism of these findings remains poorly understood, it is suggested that chronic lead exposure should be included in the differential diagnosis of unexplained intracranial calcifications in adults.

Clinical and experimental data have convincingly shown that repeated exposure to excessive amounts of inorganic lead can result in dose-related dysfunction of the central nervous system (CNS), kidneys, bone marrow, peripheral nerves, muscles, endocrine glands, and joints [1–4]. Exposure usually occurs through inhalation, although more recently, human respiratory absorption has been confirmed [1]. It has also been suggested that ingestion may become a more important route in heavy metal intoxication as air lead levels are better controlled [5]. Other possible sources of poisoning could come from retained bullets in the body, inadequately fired ceramics, indoor firing ranges, cosmetics, and herbal medicinals [6–9]. Several studies have demonstrated significantly higher incidences of neuropsychiatric manifestations in patients with known chronic lead exposure and elevated serum lead levels [1, 2, 10]. These symptoms include headache, dizziness, fatigue, depression, irritability, insomnia, and diminished libido. The nonspecificity of these complaints does not commonly alert clinicians that lead is the offending agent unless occupational risks are previously known. In our review of the literature, we found a single pathologic study that reported increased cerebellar calcification using autopsy material from patients with increased blood lead levels [11]. All of them were from Queensland, Australia, and in three patients cerebellar calcium deposits were verified by computed tomography (CT) [12]. In this report we describe three American adults who worked in a lead smelting plant for several years, had elevated serum lead levels, and were found to have subcortical, basal ganglionic, and cerebellar calcifications.

Case Reports

Case 1

A 56-year-old man had worked in a lead smelting plant for 31 years. Several serum lead levels were elevated, and on admission his level was 72 µg/dl (normal, 0–30 µg/dl). BUN,
creatinine, serum calcium, phosphorus, and parathormone values were normal. His neurologic examination revealed evidence of dementia and peripheral neuropathy. CT showed multiple punctiform and curvilinear calcifications in the subcortical area and basal ganglia (fig. 1).

Case 2

A 57-year-old man had worked in a lead smelting plant for 34 years. At one point he had a serum level of 102 μg/dl. He was advised to retire in 1979, after he developed renal disease and hypertension. On admission, he had elevated serum lead level, 66 μg/dl; BUN, 31.0 mg/dl (normal, 12–27 mg/dl); creatinine, 3.2 mg/dl (normal, 0.7–1.4 mg/dl); and parathormone, 546 pg/dl (normal, 80–375 pg/dl). Serum calcium and phosphorus were normal. Neurologic evaluation showed recent memory loss, decreased visual acuity in one eye, and diminished vibration and proprioception distally in the arms. He also complained of intermittent dizziness. CT revealed curvilinear calcifications in the subcortical region, vermis, and cerebellar hemispheres (fig. 2).
The form of hydroxyapatite [14]. When these calcium deposits reach a certain size, they can be imaged by neuroradiologic methods such as CT. In many instances, the size, distribution, and pattern of calcification observed on a brain CT scan help clinicians identify neuroanatomic landmarks and confirm their suspicion of pathologic processes.

Calcification of the cingebellum in patients with chronic lead exposure was recently demonstrated in Queensland, Australia, by postmortem studies [11]. These findings were subsequently confirmed by cerebral CT in similar patients who came from the same area [12]. Most recently, in North America, Swartz et al. [15] described cortical calcific deposits in the cerebrum in a patient who had chronic renal disease and occupational history of lead exposure. In their patient, serum lead levels were persistently within normal limits and parathormone levels were markedly elevated. They implied that chronic renal disease, increased serum calcium, and increased circulating parathormone may have led to deposition of calcium salts within the brain parenchyma. The clinical and radiographic features of our cases were different. All our patients had documented exposure to lead at work for several years and toxic lead levels in the serum. Chronic renal disease in two cases was believed to be related to chronic lead toxicity, and both patients had hypertension. In one patient neither hypertension nor renal disease was reported. Signs and symptoms of cerebrovascular disease were absent. We were the first to show basal ganglionic and curvilinear cerebral subcortical calcification in such groups of patients. We also found similar cerebellar lesions as described by Graham et al. [12].

Although CNS lesions in acute lead poisoning are well known, the mechanism of toxicity in subacute and chronic human cases is poorly understood. This is largely because the brain abnormalities seen after prolonged exposure to lead are frequently accompanied by pathologic changes in the kidneys, blood vessels, and other visceral organs. In the
series of Henderson [10], 60% of lead-intoxicated children followed longitudinally died of either renal or vascular disease. The studies of Tonge et al. [11] that showed calcium deposits around cerebellar arteries and arterioles along the horizontal fissures correlate well with the CT pattern of cerebellar calcification we described in our patients. Using histochemical techniques, they further suggested that transudation of polysaccharides across blood vessels occurs in acute lead toxicity, and the deposition of calcium salts ensues at a later stage [16]. This mechanism would also explain the cerebral subcortical and basal ganglionic calcifications in our cases. However, it is important to remember that, in certain instances, calcium deposits can be verified only by microscopy and, therefore, may not be seen by the usual radiographic technique.

It is not possible for us to relate our CT findings to any particular biochemical or local structural abnormality. Parathormone level was increased in only two cases, while the serum calcium and phosphorous values were normal in all three. The latter measurements, however, do not exclude the possibility that hypercalcemia and metastatic calcification occurred in the past. It is also difficult to suggest that calcium deposition developed in previously damaged brain parenchyma. The CT studies did not reveal other focal lesions, and neurologic deficits referable to the calcified areas were absent. To clarify these important issues, we need to follow patients with known acute and chronic lead exposure longitudinally using various neurodiagnostic methods and collect postmortem tissue for histologic and biochemical studies. However, despite the absence of an acceptable pathophysiologic mechanism, we suggest that chronic lead exposure should be considered one of the etiologic factors of intracranial calcification.

REFERENCES