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Chronic Lead Poisoning with Neuropathic presentation like Botulism: A Case Report.

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ABSTRACT

Background

Chronic lead poisoning manifestations is often non-specific and may mimic neurologic diseases, gastrointestinal problems, and hematologic disorders. When there is no history of lead exposure, it may be misdiagnosed. Trying to search occult exposure is very important and poisoning could be diagnosed by a simple test of blood lead level.

Methods

Here we describe a case of lead poisoning with presentation of acute generalized muscle weakness who was admitted with primary diagnosis of Botulism. We also reviewed the literatures on this topic.

Results

The primary diagnosis was food poisoning in further investigation, it revealed that his job is recovering of electronic piece of old computers for about 20 years. Based on recurrent uncontrollable abdominal pain, constipation, generalized weakness, associated with hypochromic microcytic anemia, a highly suspicious to chronic lead toxicity was considered. Patient's blood lead level was 149 µgr/dL (normal:2-25).

Conclusions

Detailed clinical investigation is the first step for identifying rare presentations of lead poisoning cases. The health hazards of lead poisoning are rising due to lack of education regarding the dangers of working with lead. With improving national health policies, we can prevent this chronic and subtle poisoning.

Keywords: Lead Poisoning; Neuropathy; Botulism

Introduction

Ingestion, inhalation, prenatal, and dermal exposure are the ways of human lead contamination. Chronic lead poisoning involves central and peripheral nervous system, hematologic, renal, and gastrointestinal systems. [1] Adult exposure to lead may occur in workers in various industries such as construction materials, paint, batteries and piping. [2] Manifestation of poisoning are non-specific including fatigue, anemia, reduced limb sensation, depression, reduced memory, hyperirritability and antisocial behavior. [3]

Materials and Methods

Case presentation

We present a 43 years-old male, who admitted to hospital with primary diagnosis of botulism with complaints of severe general weakness after eating un-boiled canned tuna fish, 5 days ago. He also was complaining of colic abdominal pain, constipation, generalized joint and bone pain, malaise, excess sweating, and decreased sleep, severe loss of appetite and memory loss for a few months that becoming more severe in recent week. The patient gave no history of difficulty swallowing.

On clinical examination, vital signs were normal. He seemed obviously ill and pale and had coarse tremor, without diplopia, ptosis, and dysarthria. Lungs and heart examination were normal. Further evaluation revealed that he had lost about 20 kg of weight in recent month. Forces of upper limbs were 3/5 and lower limbs were 4/5. Sensory, cerebellar and deep tendon reflexes, all were normal. Viral, metabolic, and autoimmune causes were ruled out. Imaging studies including chest radiography, upper gastrointestinal endoscopy, pelvic and abdominal sonography, and colonoscopy yielded no remarkable findings. EMG and NCV, spinal fluid examination, and non-contrast brain CT scan also were normal. So, botulism and electrolyte abnormalities and viral disease were ruled out. Table 1 shows the patient laboratory data. With regard to primary diagnosis of food poisoning a toxicologist consult was performed. In further investigation, it revealed that his job is recovering of electronic piece of old computers for about 20 years. Based on recurrent uncontrollable abdominal pain, constipation, generalized weakness, associated with hypochromic microcytic anemia, a highly suspicious to chronic lead toxicity was considered. Patient's blood lead level was 149 µgr/dL (normal: 2-25). The patient underwent dual chelation therapy with intramuscular British Anti-Lewisite (BAL) and CaNa2EDTA intravenously. In follow up visit, one month later, abdominal discomfort and extremities weakness dramatically subsided. Table 2 shows the decreasing blood lead level during treatment courses.

Table 1. Patient tests results

Tests	Value	Normal range
WBC	13000/L	4000-11000
Hb	8 g/dL	Men:13-18
MCV	74.9 fL	76-96
PLT	$353x10^9$/L	$150-400x10^9$
RDW	20.3	11.6-14.6
PT	14.5s	5-12
PTT	25s	26-38
Urea	4.9mmol/L	2.5-6.7
Creatinine	1.2mol/L	0.7-1.5
Na	137mmol/L	135-145
K	4.6mmol/L	3.5-5
AST	50iu/L	3-35
ALT	40iu/L	3-35
Alk ph	35.3iu/L	30-35
Bil (T)	1.5mg/dl	0.1-1.3
Bil (D)	0.6mg/dl	0.1-0.3
CPK	40u/l	50-200
BS	228mg/dL	100-110
Iron	109µgr/dL	60-160
Transferrine	195mg/dl	190-365
Ferritin	885g/dl	20-300
Mg	2meq/l	1.5-2
Ph	5mg/dl	3.5-5
Zn	97µmol/L	70-102
Ca	10mg/dl	8.5-10.5

Table 2. Blood lead level changes during treatment courses

	Before Chelation	After 1th course of chelation (BAL+ CaNa2EDTA)	After 2nd course of chelation (D-penicillamine)	After 3rd course of chelation (D-penicillamine)
BLL (µgr/dL)	149	73	61	55

Discussion

Since our patient's main presentation was bilateral limb weakness, we had to approach to all disease with weakness presentation. First of all, electrolyte abnormalities that can cause weakness were considered. Central and brain stem pathologies were ruled out because there was no diminution of mental status and abnormal brain CT-scan. Another differential diagnosis of limb weakness regarding to eating un-boiled canned tuna fish, was Botulism. Common symptoms of foodborne disease are difficult swallowing, blurred vision, abdominal cramps and paralysis. In this case we had none of these symptoms except distal paresthesia and Brain CT-scan, Lumbar puncture and EMG_NCV all were normal. Another suspected diagnose was Guillain-Barret syn. that manifests with bilateral ascending motor polyneuropathy. Myelopathies typically present with bilateral extremity weakness and sensory deficits. Neuropathies affect both motor & sensory symptoms with dramatically reduction of distal power. Heavy metal intoxication also was considered because of patient's occupational exposure. Lead exposure neuropathy can cause weakness with primary involving the wrists and finger extensors and it can be later spreads to other muscles. With history of occupational exposure, abdominal pain, general weakness, hypochromic microcytic anemia, and a high blood lead level, chronic lead toxicity was our net diagnosis.

Lead poisoning arises from the excess amounts of lead in serum. [4] House paints, toys, pipes & sink faucets which can contaminate drinking water are common sources of lead poisoning. It has been apparent that occupational exposure is the major cause of subtle chronic lead poisoning. The highest risk is associated with jobs that create lead dust (like powder mixing) or fume.

Manifestations of lead poisoning are sometimes non-specific including fatigue, abdominal colic, constipation, sleep problems, anemia, reduced limb sensation, depression, reduced memory, hyperirritability and antisocial behavior. [1,3] Neurological symptoms include ataxia, numbness or tingling in the extremities, joint and muscle pain, stupor, coma, convulsion, reduced IQ, Short term memory loss and Wrist/ankle drop due to axonal damage that primarily affects motor nerves. [5]

Gastrointestinal disorders include anorexia, vomiting, constipation and abdominal pain. [1] Impairment of the hematological system leads to hypochromic microcytic anemia. The renal effects of lead poisoning, consist in proximal tubular function impairment leading to aminoaciduria, glycosuria, hyperphosphaturia, and interstitial nephritis in chronic exposure and also impairment of calcium metabolism by interfering with activation of vitamin D1,2-dihydroxy cholecalciferol. [1]

Chronic exposure is associated with decline of neurocognitive function. [4] In the setting of chronic exposure, lead toxicity can be symptomatic at lower blood levels. [6] Chronic lead toxicity may cause pregnancy complications including increased number of miscarriages, low birth weight and

increased rate of preeclampsia & eclampsia. [4] In men with chronic lead exposure and blood lead levels between 40 and 70 µgr/dL, an increased percent of dysmorphic sperm was seen. [6]

So in all patients with mentioned clinical symptoms, we must evaluate occupational history, location and physical condition of patient's residence, hobbies of all family members, drinking water source and proximity to industrial facilities & hazardous waste sites.

Physical examination of patients should include special attention to the hematologic, cardiovascular, gastrointestinal, renal and nervous system. Laboratory tests that helps us for diagnosis are: Complete Blood Count, Blood lead level (the best screening and diagnostic test), Peripheral Blood Smear (PBS), Protoporphyrin level, BUN, Cr and urine analysis.

Clinical manifestations depends on individuals. Acute, subacute and chronic exposure may make distinct manifestations in each case. [7] If serum lead level exceeds 80 µgr/dL, the manifestation will be that of severe toxicity:

The first step in all suspected cases is remove the source of lead. In severe cases (with clinical symptoms or Blood Lead level >45), chelation therapy is necessary. Chelating agent binds to body lead and it excretes in urine.

Conclusion

It is concluded from the present study that detailed clinical investigation is the first step for identifying rare presentations of lead poisoning cases. The health hazards of lead poisoning are rising due to lack of education regarding the dangers of working with lead. With improving national health policies, we can prevent this chronic and subtle poisoning.

Conflicts of Interest

The authors declare no conflict of interest in this study.

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