REVISTA NEUROCIÊNCIAS

Encephalopathy after anoxic brain injury and cyanide poisoning for cassava in Brazilian Amazonia

Encefalopatia após lesão cerebral anóxica e envenenamento por cianeto por mandioca na Amazônia brasileira

Encefalopatía después de una lesión cerebral anóxica y envenenamiento por cianuro por la yuca en la Amazonía brasileña

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Resumo
O consumo de mandioca é comum na Amazônia, mas se a cassava for processada incorretamente, sua ingestão pode levar à intoxicação por cianeto com encefalopatia aguda direta ou associada às complicações clínicas. Este é o relato de um caso de encefalopatia após envenenamento por cianeto por mandioca na Amazônia brasileira. Um melhor conhecimento sobre o risco e o acesso a tratamentos específicos são ferramentas importantes para evitar esse triste resultado.
Unitermos. Mandioca; glicósidos cianogênicos; encefalopatidistonia aguda; toxina botulínica

Abstract
The consumption of cassava is a regular habit in Amazonia. But, if it is improperly processed, its ingestion may lead to cyanide intoxication with acute encephalopathy directly or associated with disabilities after clinical complications. There is case report about a encephalopathy after cyanide poisoning for cassava in Brazilian Amazonia. A better knowledge about the risk and access of specific treatment are important tools to avoid this sad outcome.
Keywords. Manihot esculenta; cyanogenic glycosides; acute encephalopathydystonia; botulinum toxin

Resumen
El consumo de yuca es común en la Amazonía, pero si la yuca se procesa incorrectamente, su ingesta puede conducir a una intoxicación por cianuro con encefalopatía aguda directa o asociada con complicaciones clínicas. Este es el informe de un caso de encefalopatía después del envenenamiento por cianuro por yuca en la Amazonía brasileña. Un mejor conocimiento sobre el riesgo y el acceso a tratamientos específicos son herramientas importantes para evitar este triste resultado.
Palabras clave. Manihot esculenta; glucósidos cianogénicos; encefalopatidistonía aguda; toxina botulínica

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INTRODUCTION

Basal ganglia injury due to intoxication, anoxic brain injury, and other many etiologies can cause movement disorders. Manioc/cassava (*Manihot esculenta Crantz*) is a traditional and indispensable kind of food in Amazonia. Cassava has high concentration of toxic cyanogenic glycoside. Ingestion of cassava without cooking can drive acute cyanide intoxication with vomiting, diarrhea, abdominal pain, acidemia, hyperlactatemia, hypotension, apnea, seizures, coma, opisthotonus, trismus, and mydriasis[1]. This case report shows an encephalopathy,in a child, due to consequences of cyanide intoxication after inappropriate preparation of cassava.

CASE REPORT

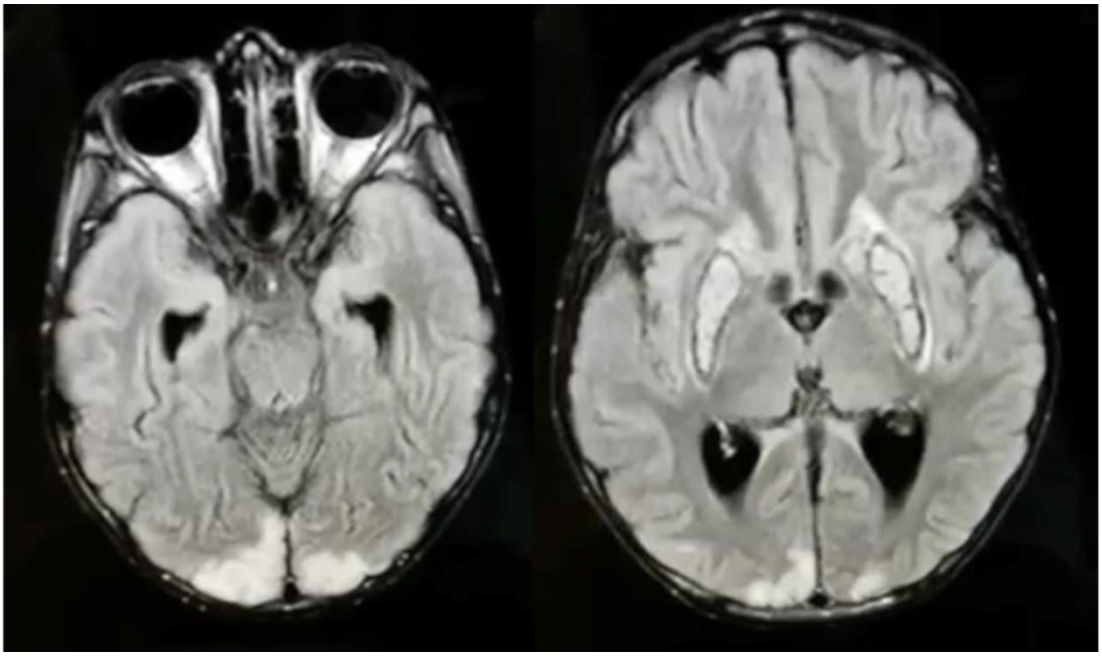
A 7-year old girl, from Amazonian rural area, ate a large amount of cassava without previous cooking. A few hours later, the patient had emesis, mental confusion, and drowsiness. She was admitted in the local primary health center with cardiac arrest with successful reversion after 15

minutes of cardiopulmonary resuscitation. After two days, in a tertiary healthy center, there was normal vital signs and nutritional status with neurological exam showing lethargy, general stiffness, hyperreflexia, bilateral Babinski sign, dystonic movements in the left side, without meningeal signs. The blood work demonstrated mild anemia (hemoglobin 10,9g%) and elevated liver enzymes. Magnetic resonance images characterized hyperintense basal ganglia and bilateral occipital lobe lesions (Figure 1). Cerebrospinal fluid had no abnormalities. After 21 days, the patient had improvement of her level of consciousness with anarthric speech, global stiffness (predominant in right side), tetraparesis (muscular strength grade 2-3), and tremor in hands. Initial therapeutic intervention (baclofen 30 mg per day; carbamazepine 400 mg per day; diazepam 15 mg per day) was unsuccessful, but botulinum toxin was applied with improvement in biceps brachii, femoral retus, medial hamstrings and gastrocnemius muscles, totaling 150 international unities.

DISCUSSION

The most common acute cyanide poisoning occurs after cyanogenic plants, herbs, and cyanide-contaminated food intake. [1]In stomach, cyanide glycosides produce highly toxic hydrogen cyanide with consequent greater toxicity[2]. Cyanide intoxication has been reported after the ingestion of apricot, cassava (specially root), cherry, and bitter almond seeds[3].

Figure 1.



Initial child's signal and symptoms were typical manifestations of cyanide poisoning[4]. Indeed, our patient had pyramidal syndrome and unusual hypertonic impairment (abnormal postures with intermittent muscle twisting, repetitive rhythmic and hyperkinetic movements). There were lesions in bilateral basal ganglia, and occipital cortex[5]. To explain all findings, an overlap of anoxic brain injury and cyanide intoxication in this case can be probable. Indeed, the consumption of cassava is a regular habit in Amazonia. If it is improperly processed, its ingestion may lead to cyanide intoxication with acute encephalopathy directly or associated with disabilities after clinical complications. There is not available data about its incidence. Populational knowledge about the risk and access of antidote are important tools to avoid this sad outcome.

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