CASE REPORTS

CIGUATERA FISH POISONING WITH ELEVATED MUSCLE ENZYMES AND ABNORMAL SPINAL MRI

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Abstract. We report three cases of ciguatera fish poisoning. One patient died secondary to respiratory failure. Two patients showed elevated muscle enzymes and one patients had an abnormal cervical spinal MRI. MRI findings have not been previously described. MRI findings explain the mechanism of the L'Hermitte phenomenon (a common complaint) among these patients. Respiratory failure is rare in ciguatera fish poisoning. Our findings suggest this could be related to respiratory muscles involvement.

INTRODUCTION

Ciguatera is the commonest marine poisoning, endemic throughout subtropical and tropical regions of the Indo-Pacific and Caribbean (Bagnis et al., 1979). Poisoning is characterized by gastrointestinal, neurological and cardiovascular symptoms (Gillespie et al., 1986). Neurological effects predominate in the Indo-Pacific region (Johnson and Jong, 1983). Neurological symptoms include paresthesia, the L'Hermitte phenomenon, myalgia and cold alldynia or “temperature reversal”, which is almost pathognomonic of ciguatera poisoning (Isbister and Kiernan, 2005). Neurological symptoms are attributed to sensory neuropathy which has been documented in some patients and may persist for long periods of time (Cameron et al., 1991b). A multiplicity of symptoms are observed in ciguatera toxicity which may be due to a variety of toxins observed with ciguatera fish poisoning. Injury to skeletal muscles with elevated muscle enzymes and MRI abnormalities have not been frequently reported in relation to ciguatera poisoning. We report three members of the same family with ciguatera toxicity, one of which died before arriving at the hospital.

A three member family (husband, age 44 years; wife, age 39 years; child, age 5 years) consumed cooked liver of an unknown type marine fish for dinner.

Case one

The wife developed symptoms of severe muscular pain over the body and electric (shock) current like feelings radiating from the neck to all extremities initially. No signs of gastrointestinal symptoms were present. Later, she developed shortness of breath, became cyanotic and expired the same day. No autopsy was performed.

Case two

The husband developed symptoms of severe muscular pain all over the body and current like feelings radiating from the neck to...
all extremities on neck movement (L'hermitte phenomenon) along with generalized weakness in the morning. His weakness was so profound that he was unable to leave home and call for help until evening. He was brought to the hospital with his dead wife and 5-year old boy by his neighbors. He was also complaining of peri oral numbness and severe myalgia. He had cold hot reversal, when his skin was washed with cold water it felt hot to him and application of hot water felt cold to him (cold allodynia). The neurological examination was normal except for motor strength of 3/5 proximally and 4/5 distally without asymmetry. The sensory examination showed generalized dysesthesia. The laboratory work-up was essentially normal except the serum creatine kinase (CK) level was elevated at 38,584 IU/l and the serum ALT (717 IU/l) and AST (595 IU/l) was also elevated. Nerve conduction studies were normal and electromyography was normal except for a few fibrils and sharp waves in the quadriceps muscles bilaterally. A MRI of the cervical spine showed a hyperintense signal involving the cervical spinal cord at the C3-4 level (Fig 1). The patient recovered completely over five weeks.

Case three

The boy also developed symptoms of pain and lower extremity weakness. A neurological examination found the child alert and oriented, the speech was weak but otherwise normal. There was generalized hypotonia and hyporeflexia with normal muscle bulk. Power was 3/5 in the lower and 4/5 in the upper limbs. The laboratory studies were essentially normal except for a CK of 4,641 IU/l and an ALT of 701 IU/l. He had a normal neurological examination on five week follow-up evaluation.

DISCUSSION

Ciguatera toxicity is commonly found in endemic regions. Clinical features in all three cases were similar suggesting ciguatera toxicity. Presence of allodynia, the L'hermitte phenomenon and paresthesias are present in 90% patients (Isbister and Kiernan, 1991). The symptoms of allodynia and paresthesia may be explained by a predominantly sensory, length dependent neuropathy which has been documented in many patients with ciguatera toxicity.

The mechanism underlying the L'hermitte phenomenon is most likely related to spinal cord involvement documented by MRI in our patient. A spinal cord lesion has not been previously described in association with ciguatera toxicity. The effect of marine toxins on voltage
gated Na+ channels in the myelin of peripheral nerves has been described (Cameron et al, 1991a). Myalgia is common in ciguatera toxicity. High CPK levels have been reported in one case. Patients with ciguatera poisoning developed acute respiratory distress and severe muscle spasms with elevated muscle enzymes. Palytoxin, isolated from fish has been identified as the responsible agent (Kodama et al, 1989). In our patient, the EMG did not show any myopathic features but muscles demonstrated some irritability. Molecular diversity of structure and function of voltage gated Na+ channels is well known (Ogata and Ohishi, 2002). We speculate that toxin leads to damage of muscle membranes through one of these voltage gated Na+ channels.

There are no laboratory tests available to confirm ingestion of fish containing ciguatoxin or palytoxin. In all three cases the common features of ciguatera toxicity were present, including presentation as a multi-system disorder with involvement of the gastrointestinal system, central and peripheral nervous system and cardiovascular system. Our findings suggest this disease may involve the spinal cord and skeletal muscles. Ciguatera poisoning patients with elevated muscle enzymes may be at risk for developing respiratory failure.

REFERENCES


