

## INTRAVENOUS LIDOCAINE FOR THE TREATMENT OF ALCOHOLIC NEUROPATHY : REPORT OF A CASE

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**SUMMARY:** *Chronic alcoholic patients are prone to nutritional deficiencies. The incidence of polyneuropathies due to chronic alcohol consumption is 30 %. We present a chronic alcoholic patient with polyneuropathy due to folic acid deficiency, aiming to propose intravenous lidocaine treatment in cases unresponsive to classical treatment.*

**Key Words :** *Polyneuropathies, Lidocaine Therapeutic Use, Alcohol Abuse.*

### INTRODUCTION

Polyneuropathies are classified as congenital or acquired polyneuropathies. The causes of acquired polyneuropathies are nutritional deficiencies, metal intoxication, drug intoxication (isoniazid, disulfiram, vincristine, paclitaxel, chloramphenicol, phenytoin, dapsone, gabapentin), metabolic diseases such as diabetes mellitus, autoimmune and infectious diseases or neoplastic/paraneoplastic diseases (1).

The nutritional factors responsible for alcoholic neuropathy are controversial. Many investigators have drawn attention to the similarities between neuropathic beriberi and alcoholic neuropathy. In 1928, Shattuck first declared that polyneuritis of chronic alcoholism is due to insufficient vitamin B intake, so that it may be a real beriberi (2). Chronic alcoholic and nutritional polyneuropathies are seen in 30 % of chronic alcohol users (3), and numbers are increasing.

Treatment strategies are cessation of alcohol, nutritional support and other analgesic interventions. Intravenous lidocaine treatment is a non-specific treatment for distal painful neuropathy. Analgesic efficacy of systemic local anesthetics has been reported for diabetic neuropathy, postherpetic neuralgia and others (4). The presented case aims to propose intravenous (IV) lidocaine treatment in a chronic alcoholic patient with folate deficiency, unresponsive to classical therapy.

### CASE REPORT

The patient was a 36-year-old man who had been a chronic alcoholic for 15 years. In January 1999, he presented with burning and numbness in both hands and feet. Psychiatric treatment for chronic alcoholism began in March 1999. He had had 20 sessions of psychotherapy, 20 sessions of alcohol conditioning treatment and 20 sessions of group therapy. He was referred to the Neurology department for his symptoms. His neurologic examination showed normal cranial nerves,

normal motor function, increased patellar and achilles reflexes. He had loss of pain and temperature sensation in his extremities in glove and sock pattern. Joint position examination and cerebellar functions were normal. Romberg test was negative. Total blood count, blood chemistry and vitamin B levels were normal except folic acid level which was 0.10 ng/ml (normal value >1.5 ng/ml). Electroneurography (ENG) showed normal motor conduction velocities in both median and ulnar nerves and peroneal and posterior tibial nerves. No sensorial compound nerve action potential (CNAP) was found in the right median nerve second finger-wrist segment. Sensorial conduction velocities were slow in palm-wrist, wrist-elbow and elbow-axilla segments. No sensorial CNAP was found in the right ulnar nerve fifth finger-wrist segment. Sensorial conduction velocities were slow in wrist-elbow and elbow-axilla segments. No sensorial CNAP was found in either sural nerves. These electrophysiological findings were interpreted as diffuse sensorial neuropathy.

Polyneuropathy treatment was started with amitriptyline HCl 10 mg/day and vitamin B complex, including folic acid twice daily. Amitriptyline dose was increased to 25 mg and then 50 mg/day weekly. The patient complained of sleeping too much, so the dose was kept at 50 mg/day. After two months, no change occurred, so the patient was referred to the Algology unit in June 1999. He had burning and throbbing pain and strain in both feet. He was able to walk with crutches and he tried not to step on his feet. He also had numbness in his hands. Pain intensity was 8 according to Visual Analog Scale (VAS). He was unresponsive to antidepressants. He did not have signs of sympathetic nervous system involvement, so epidural or sympathetic block was not performed. Opioids and sedative hypnotics were the last choice in chronic neuropathic pain, and intravenous local anesthetic treatment was planned.

5 ml/kg (260 mg) lidocaine in 100 ml of saline was infused in two hours through a forearm vein via a 20-G intravenous cannula. The patient was monitored, heart rate and pulse oximetry were followed and non-invasive blood pressure was measured every five minutes by Odam Users Manual Physiogard SM 786 1995 (France). Emergency equipment was ready at

hand. This treatment was repeated for three consequent days. VAS decreased by 40 % on the second day, 70 % on the third day. VAS was by 2 on fourth day and the patient was freely walking without crutches. Two sessions on the second week and one session on the third week were performed during the next two weeks (total six sessions). After three weeks, VAS was 1 and the patient was very comfortable. No local anesthetic toxicity signs (metallic taste, dizziness, tinnitus), allergic reactions or side effects were seen. The treatment was stopped and the patient was recalled a month later. After seven weeks, he was still symptom free. Pain and temperature sensation loss in the upper and lower extremities was still present, but it had diminished considerably. Psychiatric treatment continued.

### DISCUSSION

Neuropathic pain is caused by electrical hyperexcitability of injured sensorial neurons with abnormal impulse discharge in ectopic focuses. The source is the injured focus and accompanying dorsal root ganglion. Spontaneous discharge or mechanical (by movement) or chemical (sympathetic nervous system activation) impulses may exacerbate activity (4). Peripheral nerves have major pathological changes in polyneuropathy: Axonal degeneration, segmental demyelination and neuropathy. Axonal degeneration is more frequent in systemic, metabolic or toxic disorders. Alcohol causes subacute, symmetrical polyneuropathy.

Most polyneuropathies are painless, causing numbness and weakness only (1). Alcohol related folate deficiency is painful, as in our patient. Peripheral nerves are affected by alcohol secondary to vitamin deficiency. In fact, thiamine deficiency is seen in chronic alcohol consumption. Vitamin intake is reduced in addition to decreased gastrointestinal absorption of vitamins in chronic alcohol users. Alcoholics are prone to folic acid, pyridoxine (B6), thiamine (B1), niacine (B3) and vitamin A deficiency as these are actively transported or stored in the liver (5). Nerve, muscle and brain tissues are very susceptible to low levels of vitamins and minerals, and regression occurs in these tissues in nutritional deficiencies. Numbness begin in some fingers. Alcoholic polyneuropathy generally begins with acral and distal involvement. Painful

or burning sensation, numbness and paresthesia also accompany (1,2). Motor deficiency begins if treatment is not started immediately. Treatment is mostly symptomatic, dealing with triggering factors, diet and vitamin supplementation; also hygiene of extremities is important. Vitamin supplementation and abstinence from alcohol decrease alcoholic polyneuropathy symptoms and prevent progression of disease, but central and peripheral nerve lesions never completely recover. Nerve regeneration occurs in months. Splints are useful for stabilisation of extremities (1,4). We supported our patient with psychiatric and physical therapy measures.

Generally, tricyclic antidepressants (TAD) are the first choice in neuropathic pain. TADs inhibit reuptake of serotonin and noradrenaline. They are antagonistic to histamin,  $\mu$ -adrenergic and muscarinic cholinergic receptors. Clinical combination of TADs with mexiletine, carbamazepine or baclofen used is widespread, but controlled studies are limited (1). TADs have anticholinergic side effects; namely dry mouth, constipation, and visual disturbances. Sedation, weight gain and postural hypotension may be seen in high doses. Amitriptyline can be used at doses of 25-150 mg/day for analgesic purposes. Our patient was sedated, so we did not try incremental doses and other interventions were indicated. Aspirin or acetaminophene for hyperpathia and sympathetic block for burning pain are generally recommended. Sympathetic nervous system involvement signs, such as excessive sweating in the fingers, dorsum of the feet, palms and soles and postural hypotension are indicative of peripheral sympathetic fiber involvement (2). These were absent in our patient, therefore epidural or sympathetic block was not performed. Opioids and sedative hypnotics were the last choice in chronic neuropathic pain, and thus intravenous local anesthetic treatment was planned.

Analgesic effects of lidocaine and its role in chronic neuropathic pain syndromes are mentioned in the literature (6-8). In most pain centers, the drug is first tried intravenously. Patients are fully monitorized and intravenous lidocaine, phenytoin, phentolamine or fentanyl is infused. If pain is relieved by intravenous route the treatment is continued orally. Systemic local anesthetic administration can diminish the severity of neuropathic pain and decrease the

intensity and extent of associated allodynia at doses that do not produce symptoms of systemic toxicity (9). The antihyperalgesic effect of lidocaine is most likely the result of action on peripheral nerves (9). Local anesthetics have a membrane stabilizing effect due to sodium channel blockade and general ectopic impulse suppression (4). Lidocaine inhibits ectopic impulses from injured C afferent nociceptor fibers. If pain is relieved by 2-5 mg/kg lidocaine intravenously, oral mexiletine therapy is begun (9, 10). There are studies reporting that oral mexiletine is as effective as intravenous lidocaine treatment (5), but we did not use mexiletine because pain was relieved completely with intravenous treatment. Ferrante et al. treated thirteen patients with neuropathic pain with 500 mg lidocaine infusion over sixty minutes and concluded that analgesic response to IV lidocaine is best characterized by a precipitous "break in pain" over a narrow dosage and concentration range (11).

Intravenous lidocaine treatment has some side effects and toxicity in relation with the infusion rate and total lidocaine dose. Initial central nervous system (CNS) toxicity signs are: tinnitus, light-headedness, confusion and circumoral numbness. If these are present during treatment, infusion must be stopped for some time. Excitation events, tonic-clonic convulsions are more severe than the above-cited events, and the last step is the depression phase with unconsciousness, generalized CNS depression and respiratory events. Hypertension and tachycardia are seen during the CNS excitation phase. Myocardial depression, decreased cardiac output, and hypotension may lead to circulatory collapse (12). We did not note any side effect during lidocaine infusion (2 mg/kg in 2 hours) in our case. Pain was significantly relieved. We concluded that VAS decreased effectively with this treatment. As a result, if abstinence from alcohol, symptomatic treatment, dietary regulations and vitamin supplementation and TADs do not relieve symptoms of painful neuropathy, intravenous lidocaine treatment might be a good choice in alcoholic neuropathy.

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