Case report

A Patient with Heroin Withdrawal Presenting with Newly Onset Seizures

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Abstract
Heroin addiction needs to be considered a chronic medical illness with a serious health burden and psychosocial problems worldwide. Heroin withdrawal may manifest with a variety of clinical features ranging from simple nausea, vomiting, cramps, seizures, collapse and coma. We report a 38-year-old previously healthy man who was a heroin addict for fifteen years, presented with recurrent episodes of seizure attacks following heroin withdrawal. He did not require long term anticonvulsant treatment for seizure control

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Introduction

Heroin addiction, a serious health and social problem around the world, is associated with significant morbidity and mortality. Heroin (diacetylmorphine) which is highly addictive is derived from the seedpod of the poppy plant (*Papaver somniferum*). Heroin abuse depends on multiple factors including genetic and psychosocial factors (1). Seizures are often seen in substance abusers. It may be due to direct causes like intoxication or withdrawal and due to indirect causes such as CNS infection, stroke, cerebral trauma and metabolic derangement (2). Seizures due to heroin withdrawal are less common than in withdrawal from sedatives like barbiturates. However in critically ill patients who have been treated with sedatives and narcotics, sudden withdrawal of narcotic drugs may be a significant causative factor for newly-onset seizures (3). We report a patient with heroin withdrawal presented with newly onset seizure episodes.

Case report

A 38-year-old previously healthy male presented with four episodes of generalized tonic clonic seizures within 2 days. These episodes were associated with frothing, tongue bites and post ictal drowsiness, but no urinary or faecal incontinence was present. In between seizure attacks he regained consciousness. He did not report a history of fever, head trauma or recent alcohol or other drug abuse. He was a heroin addict for 15 years (daily usage) and had withdrawn for 4 days as he was under a rehabilitation program. On examination he had bite marks on the tongue. There was no neck stiffness or neurological deficits. Fundoscopy was normal. Pulse rate was 76 beats/min. Blood pressure was 110/70 mmHg. Other system examinations were unremarkable. Investigations showed haemoglobin of 14.5 g/dl, platelets 224×10^9/L, WBC 8.1×10^9/L with normal red cell indices. He was normoglycemic. Erythrocyte sedimentation rate (ESR) was 08 mm in first hour with C-reactive protein (CRP) 6 mg/dl. Blood and urine cultures were negative. Serum sodium, potassium, calcium and magnesium levels were 144 mmol/L, 3.8 mmol/L, 2.35 mmol/L (2.1-2.55) and 0.9 mmol/L (0.7-1.0) respectively. Serum creatinine was 1.02 mg/dl (0.9- 1.3). Blood urea level was 3.66 mmol/L. Liver function tests including liver enzymes, total protein with serum albumin and total bilirubin with fractions were normal. Coagulation profile was normal. Alkaline

phosphatase and gamma GT were within the normal ranges. Urine full report was normal. Significant changes were not detected in chest radiograph, electrocardiogram and 2D echocardiogram. Non contrast and contrast CT Brain were normal. Cerebrospinal fluid analysis was unremarkable. Electroencephalography (EEG) showed intermittent bilateral rhythmic slow wave activity in the delta range. The findings were suggestive of diffuse cortical dysfunction consistent with an encephalopathic state. But no definite epileptiform abnormality was seen. He was started on sodium valproate which was omitted after 4 days according to neurological opinion. He was discharged without drugs and advised to continue the rehabilitation program. During the follow up at medical clinic for one year, no further fits or adverse neurologic sequelae were encountered.

Discussion

Heroin is a highly addictive opioid drug which is used by millions of addicts globally. Drug or heroin addiction is a chronic, relapsing disorder. These patients have compulsive drug-seeking and drug-taking behavior. Chronic usage of heroin will cause central nervous system changes which lead to tolerance, physical dependence, sensitization, craving, and relapse (4). If the patient reduces or ceases heavy and prolong heroin use it will cause substance-specific syndrome (5). Clinical features of heroin withdrawal syndrome include muscle cramps, arthralgia, anxiety, nausea, vomiting, diarrhea, malaise, mydriasis, piloerection, diaphoresis, rhinorrhea, lacrimation, insomnia and less commonly confusion, convulsions, collapse and coma. The half-life of the opioid causing withdrawal syndrome determines the onset and duration of symptoms. In heroin withdrawal, symptoms peak in 36-72 hours and may last for 7-10 days (6)(7).

Long term activation of opioid receptor increases the activity of adenylyl cyclase, tyrosine hydroxylase and cAMP dependent protein kinase A. Also increased phosphorylation of gene transcription factors such as cAMP–responsive element–binding protein (CREB) and ΔFosB occur due to chronic activation of opioid receptors. The resultant up regulation of cAMP signaling pathways is a homeostatic response to the chronic inhibition of the locus ceruleus by opioids and is a cornerstone in withdrawal. Locus ceruleus develops tolerance to the inhibitory effect of opioids in long term exposure. When there is a withdrawal state the inhibitory effect of opioids is lost and it leads to adrenergic over activation as the firing of locus ceruleus neurons are unopposed. Then it manifests as withdrawal features. Structural changes in neurons can occur due prolonged exposure to heroin which may also contribute to withdrawal symptoms such as seizures (4)(8)(9)(10).

There are many causative factors for seizures. Mainly in adults it can be due to metabolic disorders (eg: uremia, hepatic failure, electrolyte abnormalities, hypoglycemia and hyperglycemia), illicit drug abuse, withdrawal from alcohol and drugs, trauma, brain tumors and other space occupying lesions, and infectious diseases (11)(12). In our patient other main causes were excluded. So the seizure etiology was directed towards heroin withdrawal.

Main objectives of patient care in heroin withdrawal are to relieve distress, avoid severe withdrawal manifestations, maintain proper compliance in ongoing treatment, disturb the pattern of heavy and regular drug use and assist in resolving other associated problems. Non-pharmacological therapies mainly focus on patient assessment, treatment matching and psychosocial support. One of the basic principles in managing drug withdrawal is to use an agent from the same pharmacologic class or one with a degree of cross-tolerance. Drugs used in pharmacological management are d-propoxyphene, clonidine, levo-acetyl-methadol, methadone and buprenorphine. Each drug has their own benefits and disadvantages, and due to poor efficacy and side effects some drugs have been taken out from the management (5)(7)(13). However, long-term anticonvulsant prophylaxis is usually not indicated in patients with seizures where the aetiology is suspected or diagnosed to be solely drug/ heroin withdrawal (2).

Conclusion

Even though seizures are a less common manifestation in heroin withdrawal, medical professionals need to keep in mind the possibility of developing seizures due to withdrawal as heroin use is widespread and increasing. Management of heroin withdrawal syndrome requires multidisciplinary approach including pharmacological therapy, psychosocial support, continuous monitoring and follow up. According to our knowledge, patients with heroin withdrawal presenting with newly onset seizures have not previously been reported in Sri Lanka.

Consent

Informed written consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review with the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

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