

UNUSUAL PRESENTATION OF A PATIENT WITH GBL WITHDRAWAL: A CASE REPORT

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SUMMARY

GBL (gamma-butyro-lactone) is converted to Gamma hydroxyl butyrate (GHB) in the body. GBL and GHB are available in liquid form and powder form. Once categorised under “legal highs”, these two are not associated with any dependence or withdrawal in animal studies. But there are case reports indicating their high dependence potential in humans. We here present a case of a 29 year old who came to the attention of psychiatric services with very bizarre presentation and needed a host of investigations and expert views from various medical disciplines. He was treated mainly symptomatically followed by a sudden dramatic recovery on the 11th day after presentation. GBL is getting popular as a recreational drug and its withdrawal should be seriously considered in the list of medical causes leading to Delirium.

Key words: gamma – butyro - lactone

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THE CASE:

A 29 year old Caucasian, male, single, unemployed and living with parents was admitted to the Acute Medical Care Unit on 04/04/2008.

His first contact with psychiatric services was in 1998 when he was diagnosed as suffering from depression, anxiety and panic attacks. He took a massive overdose at that time.

His second contact in 2003 was with similar presentation, when he was started on citalopram. He was later diagnosed with dysthymia. Over time he was started on Lamotrigine. There is no history of Mental Illness in the family.

Medically he has Liver disease since 2003 and was diagnosed with HIV in 2007.

He started using drugs at the age of 15. He had been on injectable heroin intermittently till Nov 2007. He had used cannabis, ketamine, flunitrazepam, nitrazepam and pain killers in the past.

He started using Gamma Butyro Lactone (GBL) around 10 years ago and continues to use it. According to him, he usually takes 2 mls at night. He says that he cannot sleep if he does not take it. Sometimes he takes more of it than usual.

When he presented himself at Accident and Emergency on 4th April, he was tremulous, sweaty, agitated, confused with a vacant stare, not speaking and had an increased muscle tone on physical examination. When he was admitted he was on Methadone 40mls morning, 20mls in the evening, Mirtazapine 30 mg once night and lamotrigine 50 mg twice daily.

At Accident and Emergency, all Blood tests were normal including his head CT scan. He then was assessed under the Mental Health Act and admitted to a psychiatric unit under section 2 of the Mental Health Act.

After admission on 4th April, he was secluded due to his extreme agitation and bizarre behaviour.

After 3 days (on 7th April) he remained confused, agitated, disinhibited, hallucinating, and restless with slurring of speech. He was transferred back to the acute medical unit. He was managed by the medical team, the psychiatric team and the Department of Genito Urinary medicine.

He was sedated with haloperidol, lorazepam and chlorpromazine.

He was assessed by a neurologist who suspected encephalitis and he was started on Acyclovir. Paul Bunnell test (Infectious Mononucleosis), tests for toxoplasmosis, Cryptococcus, AFB for Tuberculosis were all negative.

The dose of Acyclovir had to be reduced due to impaired kidney function.

A Lumbar Puncture was done under anaesthesia, an MRI was done and both were normal.

Because of his increasing CRP he was started on IV ceftriaxone and amoxicillin.

The patient continues to be highly disturbed and needed a lot of sedation. The mainstay of treatment was lorazepam and haloperidol. On 14/04/08, a plan was made for his transfer to a specialist hospital, to the HIV Unit but there was a waiting list.

His Creatinine Kinase which was normal on 04/04/08 rose to 2894 on the 14th and 3202 on the 15th April and 3567 on the 16th. The haloperidol was stopped as a cautionary measure in case he was developing Neuroleptic Malignant Syndrome.

A Lumbar Puncture and MRI repeated on 16th April were normal.

Suddenly On 17th April, he appeared much calmer, rational, oriented, started eating and drinking. He was close to normal.

He was transferred back to the psychiatric Unit on the 18th April. Everything went well thereafter and he was discharged home with an arrangement with local drug and alcohol advisory services for follow up.

DISCUSSION

There are certain drugs called "legal highs". Among them are GBL and 1,4 Butanediol (Class c drugs, December 2009) which are metabolised into GHB once ingested. GBL is a chemical solvent converted into GHB by lactamase enzyme in the blood. It is used as a recreational drug and may enhance sexual pleasure. Gamma-butyro-lactone (GBL) use has steadily grown more popular in the US and more recently in mainland Europe and UK (DrugScope 2009). GHB (banned in 2003) and GBL were new "legal highs" till recently in the market of illegal drugs. GBL became a class C drug in UK since December 2009. However, due to their easy accessibility and cheaper costs their recreational use has shown a steady increase over recent times.

Police in Wales had been warning local people about legal highs such as Ivory Wave which may contain naphyrone, mephedrone or GBL, which already have been banned (Public Health Wales 2010).

Gamma-hydroxy-butyrate (GHB) and GBL (gamma-butyro-lactone) are closely related drugs with anaesthetic and sedative effects. GHB has been a Class C drug since 2003. GBL has been made class C drug since December 2009. Both are colourless, odourless liquids and have slightly salty taste. GBL converts into GHB in the body after oral consumption (DrugScope 2009).

Although animal studies have not been any conclusive (Nicholson 2001) but there is enough clinical evidence to show that GHB use may lead to severe dependence and withdrawal (McDonough 2004).

Many patients who are GHB users are not dependent, but patients with round the clock use (eg. using GHB 2-4 hrly) may present with a difficult and confusing clinical picture (Miotto 2001) which in fact is difficult to manage. GHB and GBL produce essentially the same effects. Both drugs are basically depressants slowing down body actions. The effect of small doses is similar to having a small amount of alcohol in which case Inhibitions can be lowered and libido increased. Sleepiness, nausea, vomiting, muscle stiffness, confusion convulsions, coma and respiratory collapse can be induced by higher doses. Combination of these drugs with alcohol can be fatal (DrugScope 2009).

GHB intoxication may lead to nystagmus, ataxia, somnolence, aggression and respiratory depression. It also effects cardiovascular and gastrointestinal systems though to a lesser extent. It is the CNS depression which may lead to emergencies like coma. Vomiting is often seen in GHB toxicity (King 2005).

GBL which is found in nail polish removers, paint strippers and glue debonders and is cheaply available on

the market, as cheap as 50 p per shot and is getting prompt recognition as a new party drug (Bracci 2009).

Because of the fall in the quality of illegal street drugs, a survey, by Drug Scope Street Drug Trends Survey 2009, showed that some of the older teens and younger adult recreational drug users are swapping or combining substances including cocaine, ketamine, GHB/GBL, ecstasy, cannabis and alcohol (DrugScope 2009).

Unfortunately there is no drug test to check for GBL/GHB in urine, which complicates the matter further from the management point of view.

There is no doubt that GBL toxicity is one of the medical emergencies which needs an extensive liaison between the medical and the psychiatric teams because of the intense overlap of psychiatric symptoms, evidence of use of illicit drugs and an acute confusional state. A high degree of suspicion is required when considering delirium in a patient who has a history of illicit drug use.

There are no specified medications or any treatment regimen to avoid/control withdrawals from GBL though Benzodiazepines are the best symptomatic drugs available and large quantities may need to be used at times (use of chlordiazepoxide in alcohol withdrawal signifies the similarities between GBL and alcohol). Withdrawal may last for 7-10 days so the treating doctors need to be aware, have patience, intensive monitoring of the patient and a symptomatic management usually requiring multiple staff involvement.

One of the major problems in its management is that even if the diagnosis of GBL/GHB withdrawal is clear, it still raise a much anxiety in the treating doctors, to rule out any other serious illnesses like meningitis /encephalitis which can also be fatal though much more quickly if left untreated. GHB/GBL withdrawal cases can present with late death. One case of GHB withdrawal had an unexpected death on day 12 (Dyer 2001). Because the withdrawal symptoms are really severe and appear organic until and unless proven otherwise, it is essential that the patient be nursed in an acute medical setting rather than in a psychiatric hospital and at least for 10-14 days. Quality of day to day management can be greatly enhanced by involving psychiatric inpatient nurses as expertise will be required to manage a high level of irritability, agitation and aggression resulting from the confusion.

Sudden recovery occurs and is really surprising in most of the cases.

CONCLUSION AND RECOMMENDATIONS

- GBL/GHB withdrawal continues to be a difficult condition to recognise due to non availability of any test for its detection in the blood/urine. It is high time in this new era of "legal highs" to develop a test to detect GBL/GBH consumption.

- GBL/GHB withdrawal has a very similar presentation to Alcohol Withdrawal and the mainstay of treatment is judicious use of benzodiazepines as in Alcohol Withdrawal.
- GBL/GHB withdrawal is a medical emergency and is mainly a diagnosis by exclusion.
- As one drug is banned another drug is thrown into the drug market under the name of “legal Highs”, so a high level of caution and a strict legal monitoring is needed as no one knows what someone is consuming under the name of “legal highs”.

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