Cases of buprenorphine abuse in India


Buprenorphine was introduced as a potent analgesic with low abuse potential. Reports of buprenorphine abuse by opiate abusers have accumulated over the years, highlighting its use as a cheap alternative to heroin. The lower potency compared with heroin is being compensated by using a cocktail of buprenorphine with benzodiazepines or cyclizine. This study of 18 cases seen over 3 years broadly confirms these findings. Four cases reported haematemesis during acute withdrawal, a symptom not reported in earlier studies.

Buprenorphine is a mixed agonist-antagonist opioid available in sublingual, oral and parenteral preparations. As an analgesic it is 25 to 40 times more potent than morphine (1). Nevertheless, it has a lower overdose lethality (2), milder euphoric effects and mild and delayed withdrawal symptoms (3,4). Hence, it was claimed to be a safe opiate analgesic with low abuse potential (3).

Challenging this claim, the first report of buprenorphine abuse came from New Zealand (5). In subsequent reports, abuse by the oral as well as parenteral routes was implicated (6-17) and the concurrent abuse of cyclizine or temazepam (12-17) purportedly enhanced the euphoriant effect of buprenorphine (15-17). Almost all the studies reported buprenorphine abuse as a substitute among opiate, mainly heroin, addicts.

In India, buprenorphine became available in 1986 as a prescription drug. From 1987 to 1990, our centre has recorded a gradual increase in the number of cases of buprenorphine dependence. All cases were using buprenorphine intravenously. An initial report of 3 cases, the first such report from India, has already been published (9). This study deals with findings among all the buprenorphine cases seen so far.

Material and methods

The Department of Psychiatry at the Postgraduate Institute of Medical Education and Research, Chandigarh, India, has been running a de-addiction clinic since 1978. The clinic was upgraded to a de-addiction and treatment centre in 1988 under the drug control programme of the Government of India. The centre receives the cases on the basis of self, family or case-to-case referral or from other medical or non-medical agencies. The cases are first seen in the general psychiatric clinic, which gives them an appointment for the de-addiction clinic and medical attention in the intervening period of 1–2 weeks. The de-addiction centre services include outpatient and inpatient programmes of detoxification and psychosocial intervention by a team of psychiatrists, clinical psychologists and social workers. The follow-up comprises regular outpatient visits and, in case of a missed appointment, 2 call letters and/or a home visit by a social worker for the local city patients.

Over the last decade we have registered a gradual increase in the cases of dependence on heroin and other synthetic opiates. The first case of buprenorphine dependence was registered in 1987. This report is based on 18 cases of buprenorphine dependence (of 107 cases of opiate dependence) seen between January 1987 and April 1990. All the 18 cases were abusing buprenorphine intravenously and in 11 cases buprenorphine was being used as a buprenorphine-diazepam cocktail. All the cases were re-examined by either of the first two authors to obtain detailed information about the drug abuse. Urine analysis for opiates could not be carried out, as the facilities were not available.

Results

All 18 cases were men from 19 to 37 years old (mean age 26 years); half were married and other half unmarried; 16 were from urban areas; 14 cases had completed school; 9 cases had never held any occupation; all the 9 employed cases had a middle or lower occupational status; the head of the family
was the father in 12 cases, mother in 2 cases and self in 4 cases.

All the cases, except one, were abusing other opioids and/or other drugs before abusing buprenorphine. Concurrent abuse was mainly of alcohol and cannabis, while the common substances of past abuse were heroin, cannabis, alcohol and opium.

The total duration of opioid abuse varied from 2 to 12 years (mean 5 years); the duration of buprenorphine abuse varied from 4 to 36 months (mean 14 months). The daily doses varied from 1 to 7 mg (mean 3 mg) for buprenorphine and from 30 to 100 mg (mean 60 mg) for diazepam. The common pattern of usage was 3 to 4 intravenous doses daily of 0.6 mg buprenorphine, with or without 10 to 20 mg diazepam, self-injected at 4- to 8-h intervals. Although 4 cases were using the drug in group setting only, others were using the drug either in group setting or when alone. All cases were using disposable needle-syringe sets, each set being used for 3 to 6 days, rinsed with tap-water after each use. Ten cases reported having shared their syringes or needles with their group-mates at some time or the other.

Of 3 concurrent heroin users, 2 reported much less euphoria with heroin than with the period of heroin use before abusing buprenorphine. The use of buprenorphine had been picked up from the fellow-addicts (n = 12) and medical practitioners (n = 6). The reasons for starting buprenorphine were non-availability of heroin (n = 10), to decrease the intake of heroin (n = 14) and low cost (n = 3). The daily expenses on buprenorphine (with or without diazepam) abuse were 4 to 6 times less than for heroin abuse.

The buprenorphine-diazepam cocktail was described to be more enjoyable than buprenorphine alone in terms of the "kick" being more intense (n = 4) and more rapid in onset or longer lasting or both (n = 7). The cocktail effects were reported to last for 45–180 min vs 30–45 min for buprenorphine alone.

Aches and pains, insomnia, nasal symptoms, irritability and restlessness were the most frequent withdrawal symptoms reported; muscle twitching, diarrhoea and palpitation were least frequent.

The general reporting of withdrawal severity was “50% milder than heroin”. The withdrawal was reported (and also observed in 9 hospitalized cases) to start 1–2 days after the last dose, peak at 2 to 3 days and subside by 15–20 days, except for aches and pains, which lasted for 3–6 weeks. A remarkable symptom reported during the acute withdrawal in 4 cases was haematemesis; 2–5 ml of fresh blood, up to 3 times a day. In 2 such hospitalized cases the endoscopic examination revealed gastric antral erosions.

The detoxification was carried out in the outpa-

tient department and in the wards (9 cases each). In all cases the patient and/or the family were given counselling about drug and opiate abuse and its management, including guidance about such specific problems as interpersonal or occupational difficulties. Nine cases were detoxified with clonidine; others were detoxified under substitution with meperidine or morphine.

The follow-up data are based on regular appointments and special appointment through a call letter, for the purpose of this study. The details of the follow-up showed that about three fourths of the cases were lost by 3 visits or by 1 year. The drug abuse status at the last follow-up visit showed that 8 cases were abstaining and 10 cases had restarted buprenorphine or heroin (5 cases each). Of the 5 cases restarting buprenorphine, compared with pre-treatment daily dose, 2 cases were using lower doses and 3 cases were using the same or higher doses.

Discussion

Compared with Ireland, where buprenorphine abuse was reported about 6 years after its introduction (16), in India the lag period was only 1 year. However, the increase in the number of abusers among the opiate abusers was not as rapid at our centre. This may be due to the overall lower prevalence of abusers of hard drugs or more stringent legal provision against opiates.

Almost all cases graduated to buprenorphine from heroin and 14 of 17 cases shifted either due to non-availability of heroin or to decrease the heroin consumption, confirming the earlier reports that buprenorphine is abused not as the preferred drug but as an alternative to heroin. The preference of the buprenorphine-diazepam cocktail abuser for the cocktail over buprenorphine alone confirms the earlier subjective patient reports that buprenorphine has a low euphoriant effect and that cocktail with temazepam or cyclizine enhances this effect (15–17). The preference for intramuscular route reported by the 2 other Indian studies (10, 11) is in contrast to our findings and cannot be explained. The partial opiate-antagonistic effect of buprenorphine is confirmed by some though not all of the cases reporting decreased euphoria with concurrently used heroin.

That all the cases, except for one, had started the drug abuse career with drugs other than buprenorphine suggests its low prescription in general practice. This is also supported by the fact that all the 6 cases initiated to buprenorphine by the medical practitioners were exposed to substitution withdrawal therapy for opiate dependence. The same was true for all fellow addicts who initiated some of our cases to buprenorphine.
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Two strong reasons for the continued use of buprenorphine seem to be its availability through legal channels and its being cheaper than heroin (16). The withdrawal symptoms, as reported and as observed in the hospitalized cases, were milder than after heroin. But the onset of withdrawal syndrome was not delayed by about 2 weeks, as expected according to pharmacological studies (3,4).

In 9 hospitalized cases the laboratory investigations (haemogram, urine-routine examination, liver function tests and human immunodeficiency virus test) revealed no abnormality. An occasional and mild haematemesis as a part of withdrawal syndromes has not been reported earlier. In our cases reporting haematemesis, the history and examination suggested no other physical pathology to explain the symptom.

The follow-up shows generally poor outcome and an early drop-out, though the cases followed-up for more than 6 months were more often abstaining or had reduced the intake. Of the 10 cases restarting opiate-abuse, 5 had reverted back to heroin, indicating the greater euphoriant and abuse potential of heroin.

References