Clonidine Abuse – Risk in the Psychiatric Population?

Clonidine is not generally regarded as a primary drug of abuse [1]. In rats clonidine proved to have some reinforcing effects, although primary physical dependence was not demonstrated [2]. However, there has lately been an increasing number of reports on clonidine abuse in polydrug users [3-5]. These reports deserve careful attention on the background of widespread use of clonidine for psychiatric disorders, e.g. childhood hyperactivity, nicotine, alcohol and opiate withdrawal syndromes. The stated reasons for abuse were to ‘boost’ the effects of simultaneously ingested opiates or benzodiazepines, to abate withdrawal symptoms and for its sedative or euphoric properties [3, 4].

We recently were confronted with a 52-year-old woman complaining of restlessness, agitation, anxiety and visual hallucinations. She admitted to abuse of diazepam (40-60 mg daily) in combination with clonidine (average dose between 2 and 4 mg daily) but no other concomitant medication over the past 6 years. Previously she had been treated for a supposed depressive illness with various antide-pressants, neuroleptics and amphetamines. Detailed social history included occasional smoking, but no use of alcohol. In contrast to previous reports on clonidine abuse the patient had never ingested opiates. She claimed that administration of clonidine led to an elevation in her mood coupled with a greater sense of achievement. The patient asked to withdraw from both drugs. Initially she received 0.9 mg of clonidine daily in combination with 30 mg diazepam. The latter could be discontinued in the course of 1 week with little discomfort except for the presence of nervousness. On day 4 after discontinuation of diazepam we started reducing the dose of clonidine by 0.15 mg every 4th day down to a daily dose of 3 × 0.15 mg. This was complicated by severe ‘rebound hypertension’ (peak pressures 240/130 mm Hg) difficult to manage despite treatment with calcium antagonists, diuretics and ACE inhibitors. Hypertensive episodes were accompanied by tachycardias, anxiety, excessive sweating, insomnia and visual hallucinations. Although reinitiation of clonidine 0.3 mg was rapidly followed by subjective and objective clinical improvement, it might also be possible that in our patient simultaneous withdrawal from both drugs, diazepam and clonidine, resulted in these exaggerated symptoms. Severity of withdrawal symptoms requiring close monitoring allowed a much slower decrease of clonidine than intended, so that an additional 9 weeks’ hospital stay was necessary which increased the hospital charges by US$ 17,000.

Clonidine’s side effects including significant influence on central nervous system function require its judicious use in the psychiatric population. Because of a potential danger of abuse we suggest utmost caution should prevail concerning outpatient treatment with clonidine in
depressive or suicidal patients as well as in those with the potential for illicit drug use. Clonidine use/abuse should probably receive a closer look in patients with psychiatric disorders.

References