Buprenorphine-associated gastroparesis during in-patient heroin detoxification

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ABSTRACT

Background Buprenorphine is a partial mu receptor agonist used in opiate detoxification. It has been shown to cause delayed gastric emptying in healthy volunteers. Case description We describe a case of clinically severe gastroparesis (delayed gastric emptying due to impaired contraction of the stomach) whose onset coincided with the commencement of buprenorphine-assisted detoxification. Conclusion We review the literature on gastric effects of buprenorphine in healthy volunteers, providing proof of the concept that this was the most probable cause of this patient’s gastroparesis.

Keywords Buprenorphine, gastroparesis, in-patient, opiate detoxification.

INTRODUCTION

Gastroparesis is the condition of delayed gastric emptying, which may be part of an extensive gastrointestinal motility disorder or may be isolated to the stomach. The mechanism is due usually to neurological inhibition via disruption of gut motor activity (traumatic, surgical, neuropathic/diabetic, pharmacological, infective) or myopathic disorders (scleroderma, infiltration). Opiates are known to cause constipation and ileus but are less well described as a cause of isolated gastroparesis.

CASE REPORT

In September of 2004, a 24-year-old Vietnamese-born female was admitted for in-patient detoxification for heroin dependence. She had been injecting heroin regularly for 4 years and had undergone previous buprenorphine-assisted heroin detoxification in 2002 without complication. During the current admission she was treated with sublingual buprenorphine with a standard induction regimen. When judged as being in opioid withdrawal (Clinical Institute Narcotic Assessment scale), she was given 4 mg of buprenorphine sublingually. After an hour she developed persistent nausea and vomiting; after 4 hours she was given a further 2 mg of buprenorphine without apparent benefit. Her pupils were mid-sized without suggestion of opiate intoxication. She refused further treatment with buprenorphine or any other medication. She discharged herself from hospital after 72 hours of supportive treatment without resolution of her vomiting.

She re-presented within 1 day of discharge to the emergency department because of intractable vomiting, having injected heroin in the intervening 24 hours. A plain abdominal film showed a large gastric bubble with an air-fluid level. Computerized tomography scanning revealed a massively distended stomach. A nasogastric tube was inserted. A gastrograffin follow-through showed hold-up in the stomach without mechanical obstruction and delayed transit into the duodenum. Gastroscopy showed a fluid-filled aperistaltic stomach without mechanical obstruction. Biopsies showed chronic active gastritis without Helicobacter pylori being seen. The Gastric Emptying Study showed a t1/2 of 247 minutes (normal < 90 minutes).

Clinically there was no evidence of a connective tissue disease, an eating disorder or diabetes; a fasting serum glucose was normal. She drinks no alcohol and is hepatitis C antibody-positive with a normal serum liver chemistry profile. Although atrophic gastritis has been recognized as a cause of gastroparesis it is usually only manifest with solids and her temporal relationship with the use of buprenorphine makes this drug far more likely to be the cause of her disease.
She was treated with metoclopramide with some improvement and again discharged herself from hospital against medical advice. Some months later, and on two further occasions, the patient was readmitted for detoxification without clinical evidence of gastroparesis. On all subsequent admissions she has refused treatment with buprenorphine.

**DISCUSSION**

Buprenorphine is a partial mu opiate agonist and a kappa receptor antagonist [1]. It has a high affinity for the mu receptor in the central nervous system. It is 25–50 times more potent than morphine and is used in many countries for analgesia. Given sublingually, its breakdown via first-pass metabolism is prevented. Its prolonged duration of effects because of enterohepatic cycling lends itself to be used for opiate detoxification by preventing withdrawal symptoms. Buprenorphine’s efficacy for detoxification of opiate dependence has been proven [2–4]. It is known to cause ileus and constipation but has not been described previously to cause clinical gastroparesis. Two studies have looked specifically at buprenorphine’s effect on gastric emptying [5,6]. Both studies found prolonged gastric emptying in healthy volunteers, providing proof of the concept that buprenorphine was the probable cause of this patient’s gastroparesis. Heroin is another possible causative agent, but her symptoms were present prior to her relapse to further heroin use. Furthermore, she had been using heroin for several years prior to this episode and she continues to use heroin currently. There has been no evidence of the gastroparesis during at least three subsequent admissions to our detoxification unit.

It is not clear how long the gastroparesis takes to recover but, as with other opioids, it is likely to be reversible on cessation of the medication.

**References**