Acute Gastroduodenal Perforations Associated with Use of Crack

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Crack, the free-base form of cocaine, was introduced as an illicit street drug in 1986. Since then, we have noted a significant increase in acute gastroduodenal perforations. Between 1982 and 1986, we treated 11 patients with such perforations. This represents a constant occurrence rate of 6% of hospital admissions for peptic ulcer disease. Since 1986 we have treated 16 patients with gastroduodenal perforation, which yields an occurrence rate of 16%. Nine of the 16 patients had a close temporal relationship between the use of crack and the onset of their perforation. This group was younger and disproportionately comprised of male patients. These findings led us to believe that there may be a pathogenic relationship between the use of crack and acute gastroduodenal perforation, and the clinician should be aware of the various potential complications of this new drug. This relationship also raises questions about the exact pathophysiology of peptic ulcer disease.

COCAINE IS AN alkaloid prepared from the leaves of the *Erythroxylon coca* plant. It acts at the synaptic level where it alters neurotransmitter reuptake, potentiating the effects of norepinephrine and dopamine.[1] Crack, the new free-base form of cocaine that has grown in popularity throughout the major metropolitan areas of the United States, is not destroyed by heating and can therefore be effectively administered by smoking. Many well-documented pulmonary, cardiac, obstetric, neurologic, and gastrointestinal complications have been associated with cocaine use.[1-4] The advent of crack as a popular street drug has recently introduced additional complications[5-12] (Table 1). A frequent association between gastroduodenal perforations and a history of crack use seen at our institution during the past 2 years prompted a review of all gastroduodenal perforations since 1982.

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Methods

All hospital admissions for peptic ulcer disease between 1982 and 1988 were reviewed. Twenty-seven consecutive cases of nonmalignant, nontraumatic gastroduodenal perforations were identified and reviewed for age, sex, antecedent drug use, previous medical or surgical treatment of peptic ulcer disease and operative findings, and management of the perforation.

Results

There were 27 cases of gastroduodenal perforations in our institution from 1982 to 1988. In nine cases there was an association with crack use (group I). The remaining 18 patients had no history of crack use (group II). Diagnosis of perforation was based on a combination of careful history, physical examination, marked leukocytosis, and free air under the diaphragm on a chest radiograph.

There were eight men and one woman in group I. They all presented between 1986 and 1988. Their average age was 30 years, ranging from 22 to 48 years. Two of the nine patients had a previous history of peptic ulcer disease. The time interval between the perforation (as suggested by the characteristic acute onset of sharp epigastric pain) and the smoking of crack ranged from one hour to three days (Table 2). All perforations were either juxtapyloric or within the first part of the duodenum. In the two cases with a history of peptic ulcer disease, chronic ulcerations were found. There was gross spillage of gastric contents in all cases, and a pelvic abscess in one. Intraoperative cultures revealed combinations of *Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus aeruginosa, Proteus mirabilis,* and *Candida albicans.* All nine patients underwent exploratory laparotomy with Roscoe-Graham omentopexy. There were no perioperative deaths; the av-

TABLE 1. *Complications of Crack Use*

Author	Complication	Age	Sex
Shesser,[5] 1985	Pneumomediastinum/ pneumothorax	26	F
Shesser,[5] 1985	Pneumothorax	19	F
Patel,[6] 1987	Bronchiolitis obliterans pneumonia	32	M
Murray,[7] 1988	Diffuse alveolar hemorrhage	36	F
Morris,[8] 1985	Pneumomediastinum	21	M
Zamora-Quezada,[11] 1988	Diffuse muscle and skin necrosis	20	F
Androuny,[12] 1985	Pneumopericardium	20	M

TABLE 3. *Characteristics of Patients with Gastroduodenal Perforation*

Characteristic	Group I (crack)	Group II (no crack)
Age (years)	30	44
Sex ratio		
Male (%)	89	61
Female (%)	11	39
History of PUD (%)	22	61
Treatment (%)		
Omentopexy	100	94
Antrectomy/vagotomy		6

PUD, peptic ulcer disease.

erage length of stay was nine days. All patients were placed on H-2 blocker and antacid therapy at time of discharge.

Group II consisted of 18 patients, none of whom gave a history of crack use. The average age for this group was 44 years. The male-to-female ratio was 11:7. Group II patients represent 6% of all patients admitted for peptic ulcer disease, averaging three perforations per year. Eleven patients had a history of peptic ulcer disease, nine of whom were being treated with antacids and H-2 blockers. None of the patients had undergone previous surgery for their disease. All patients underwent immediate abdominal exploration. Roscoe-Graham omentopexy was performed in 17 cases and antrectomy and vagotomy in one. There was one perioperative death in this group. Table 3 summarizes the characteristics of both groups.

Discussion

Within the last 2 years, we have seen a dramatic increase in the incidence of acute gastroduodenal perforations (Fig. 1). Two groups of patients can be distinguished, differing in mean age and male:female ratio. Group II appears to be representative of the general population of patients with a perforated peptic ulcer with regard to incidence, age, and sex distribution. Perforated peptic ulcers account for 5% to 10% percent of all hospital admissions related to peptic ulcer disease.[13-17] Jordan et al.[13] have shown that, despite a decline in the overall incidence of peptic ulcer disease during the past decades, the number of cases of perforated peptic ulcers has not significantly changed. There has been an increase in perforations in the older age group with a shift in peak age from 40 to 49 years.[13] The male-to-female ratio for peptic ulcer disease has declined from 2.2:1 in 1965 to 1.2:1 in 1981.[16]

In contrast to this group of predictable perforations, we have observed the appearance, since 1986, of a rapidly enlarging group of patients (group I) who are relatively younger, almost always male, and who all give a history of crack use closely related to the time of gastroduodenal perforation (Fig. 2). These patients account for the overall increase in incidence of gastroduodenal perforations, from 6% to 16% of all patients admitted for peptic ulcer disease.

Crack, the free-base form of cocaine, is now readily available as a street drug. Although its different form may uncover complications not previously seen, the pharmacologic properties of crack appear to be similar to cocaine,[1] namely an alteration of neurotransmitter reuptake. At the gastrointestinal level, this potentiating effect on norepinephrine results in an intense vasoconstriction secondary to stimulation of alpha-adrenergic receptors in the mesenteric vasculature, leading to focal tissue ischemia and perforation.[1-3] The chronologic relationship of crack use to gastroduodenal perforation leads us to surmise that a possible crack-related ischemic event is the cause of per-

TABLE 2. *Gastroduodenal Perforations Associated with Crack Use*

Age	Sex	History of Crack Use	PUD
26	M	48 hours before	No
33	F	6 hours before	Yes
31	M	8 hours before	No
32	M	8 hours before	No
22	M	4 hours before	No
25	M	24 hours before	No
29	M	8 hours before	No
48	M	60 hours before*	Yes
30	M	1 hour before	No

PUD, peptic ulcer disease.

* Delayed presentation. However perforation is estimated to occur less than 24 hours after crack use.

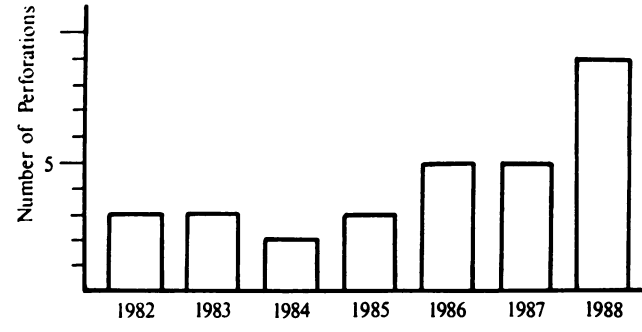

FIG. 1. Incidence of gastroduodenal perforations at St. Mary's hospital, 1982 to 1988.

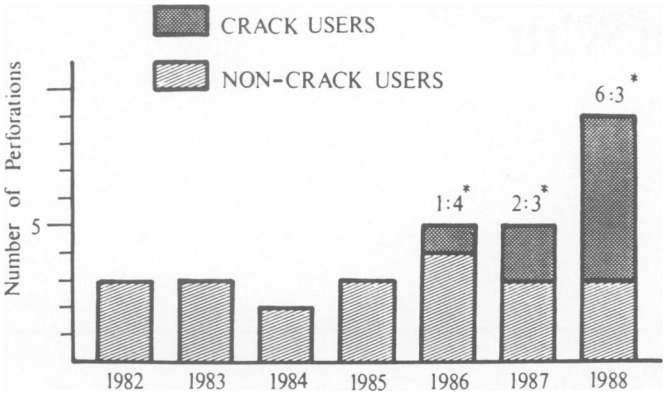

FIG. 2. Incidence of gastroduodenal perforations and its association with the use of crack.
* Crack:No crack ratio.

foration in these patients. For postoperative therapy these patients should probably receive H-2 blockers and/or antacids until it can be clearly demonstrated that they are not at an increased risk for the development of peptic ulcer disease.

We report a preliminary series of nine patients treated at our institution during the past 2 years for acute gastroduodenal perforations chronologically associated with the use of crack. Although the exact pathophysiology is not known, we believe that this new form of cocaine may be contributing to, if not inducing, these perforations. Physicians in urban areas, where this recreational drug is rapidly becoming more popular, should be aware of this and possibly other complications of crack use.

Comment

The relationship of crack use, focal tissue ischemia, and gastroduodenal perforation raises fundamental questions about the etiologies of peptic ulcer disease. One third of the patients who present with an acute gastroduodenal perforation without previous history of peptic ulcer disease will heal completely without further treatment, after surgical management of the acute perforation,[17,18] leaving one to wonder if these patients have a bonafide ulcer diathesis. Indeed they may only have had an acute ischemic episode resulting in the acute perforation.

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