

The Epidemic of Cocaine-Related Juxtapyloric Perforations

With a Comment on the Importance of Testing for *Helicobacter pylori*

David V. Feliciano, MD, FACS, John C. Ojukwu, MD, Grace S. Rozycki, MD, FACS, Robert B. Ballard, MD, Walter L. Ingram, MD, Jeffrey Salomone, MD, Nicholas Namias, MD, and Paul G. Newman, MD

From the Department of Surgery, Emory University School of Medicine and Grady Memorial Hospital, Atlanta, Georgia

Objective

This is a report of 50 consecutive patients with juxtapyloric perforations after smoking “crack” cocaine (cocaine base) at one urban public hospital.

Summary Background Data

Although the exact causal relation between smoking crack cocaine and a subsequent juxtapyloric perforation has not been defined, surgical services in urban public hospitals now treat significant numbers of male addicts with such perforations. This report describes the patient set, presentation, and surgical management and suggests a possible role for *Helicobacter pylori* in contributing to these perforations.

Methods

A retrospective chart review was performed, supplemented by data from the patient log in the department of surgery.

Results

From 1994 to 1998, 50 consecutive patients (48 men, 2 women) with a mean age of 37 had epigastric pain and signs of peritonitis a median of 2 to 4 hours (but up to 48 hours) after smoking crack cocaine. A history of chronic smoking of crack as well as chronic alcohol abuse was noted in all patients; four had a prior history of presumed ulcer disease in

the upper gastrointestinal tract. Free air was present on an upright abdominal x-ray in 84% of patients, and all underwent operative management. A 3- to 5-mm juxtapyloric perforation, usually in the prepyloric area, was found in all patients. Omental patch closure was used in 49 patients and falciform ligament closure in 1. Two patients underwent parietal cell vagotomy as well.

In the later period of the review, antral mucosal biopsies were performed through the juxtapyloric perforation in five patients. Urease testing was positive for infection with *H. pylori* in four, and these patients were prescribed appropriate antimicrobial drugs.

Conclusions

Juxtapyloric perforations after the smoking of crack cocaine occur in a largely male population of drug addicts who are 8 to 10 years younger than the patient group that historically has perforations in the pyloroduodenal area. These perforations are usually 3 to 5 mm in diameter, and an antral mucosal biopsy for subsequent urease testing should be performed if the location and size of the ulcer allow this to be done safely. Omental patch closure is appropriate therapy for patients without a history of prior ulcer disease; antimicrobial therapy and omeprazole are prescribed when *H. pylori* is present.

In 1996, an estimated 1.7 million persons in the United States used cocaine (benzoylecgonine or $C_{17}H_{21}NO_4$)

at least once a month.¹ Approximately 40% of these addicts used “crack” cocaine (cocaine base), a street product made by mixing cocaine hydrochloride (powder or crystalline cocaine) with alkali in boiling water.

Crack cocaine has been available for illicit use for approximately 12 years in the United States. Although the numerous adverse effects of using cocaine hydrochloride intranasally (“snorting”) or intravenously are well known,^{2–11} only a few

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reports have documented the complication of gastroduodenal perforation after the smoking of crack cocaine in glass pipes or cigarettes.¹²⁻¹⁶ This is a report of the recent experience with cocaine-related juxtapyloric gastrointestinal perforations at Grady Memorial Hospital in Atlanta, Georgia.

METHODS

Data were obtained by a retrospective chart review of all patients undergoing emergency laparotomies for cocaine-related juxtapyloric gastrointestinal perforations by surgical faculty and residents from Emory University at Grady Memorial Hospital, Atlanta, Georgia. Additional data were obtained from a patient log maintained in the department of surgery.

RESULTS

From 1994 to 1998, 50 consecutive patients (48 men, 2 women) with a mean age of 37 years (range 24 to 52) were treated for acute juxtapyloric gastrointestinal perforations after smoking crack cocaine. During the same time interval at the same hospital, a total of 140 patients with acute gastroduodenal perforations underwent emergency surgery; therefore, perforations related to smoking crack cocaine accounted for 35.7% of the total. The median time of onset of abdominal pain was 2 to 4 hours after smoking crack, although it was nearly 48 hours in one patient. All 50 patients were crack addicts, all were chronic abusers of alcohol, and 4 had some prior history of ulcer disease. Epigastric pain was present in 48 patients (96%), signs of peritonitis were noted on physical examination in all 50 patients, and free intraperitoneal air was present on an upright abdominal x-ray in 42 patients (84%).

At the time of laparotomy, all perforations (median size 3 to 5 mm) were in the prepyloric or juxtapyloric area or in the first portion of the duodenum. The majority, however, were in the prepyloric or juxtapyloric area.

All patients underwent exploratory laparotomy. Surgical management included closure of the perforated ulcer with a viable omental patch in 49 patients and with the falciform ligament in 1 patient. In two of the patients with a prior history of ulcer disease and only modest peritonitis at the time of laparotomy, a parietal cell vagotomy was performed as well. In the last several months of the series, antral mucosal biopsies were performed by enlarging the site of the perforated ulcer in five patients. After placing a fragment of the specimen in a CLOtest (Tri-Med Specialties, Inc., Western Australia), the urease enzyme of *Helicobacter pylori* was found to be present in four of these patients.

No intraabdominal abscesses occurred after surgery. Reflecting a policy of mandatory presence of a nasogastric tube and NPO status for 5 to 6 days after omental patch closure of a perforated gastroduodenal ulcer, the mean length of stay was 8 days. The four patients who had

positive test results for the presence of *H. pylori* were prescribed appropriate antimicrobials and omeprazole.

Outpatient follow-up was documented in <50% of patients. No effort was made to obtain long-term follow-up in this group of patients.

DISCUSSION

Although the incidence of perforated duodenal ulcers in the United States has been reported to be decreasing, patients with this problem continue to be seen at public hospitals. During the past 5 to 10 years, a significant percentage of patients with acute juxtapyloric perforations treated in such hospitals have been young men who have recently inhaled crack cocaine.

There are several reasons for the continuing use of crack cocaine among chronic users of illicit drugs. First, the 100-mg (or more) "rock" (small cube of crack) sold by the dealer on the street is 75% to 90% pure cocaine, costs only \$5 to \$10, and is easy to carry and hide from the police.² Second, crack can be smoked directly by placing part of a rock on a small screen in the bowl of a glass pipe and heating the bowl externally with a cigarette lighter. As the crack vaporizes from the heat and a crackling sound is heard, inhalation of the vapors will produce an effect in the brain in <20 seconds.¹⁰ This ease of use and the rapid effect, without the risks of intravenous injection as was practiced with cocaine hydrochloride, make crack a most appealing street drug. Third, crack has a significant potential for rapidly leading to chronic addiction, as with all types of cocaine. In the report by Washton et al,² 144 crack addicts were asked to rate how addicting crack is on a scale of 0 (not addictive) to 10 (extremely addictive). A rating of 8 or higher was chosen by 88% of those surveyed. This certainly helps explain why >38% of the 333,359 patients entering publicly funded drug treatment programs in the United States in 1995 listed cocaine as their primary illicit drug.¹⁷ Fourth, there is the well-known pharmacologic phenomenon that occurs when the abuser of cocaine simultaneously abuses alcohol. The human liver combines these agents into cocaethylene, a substance that intensifies the euphoric effect of cocaine.¹⁸

When given systemically, cocaine inhibits the presynaptic reuptake of dopamine and increases the postsynaptic concentration of this neurotransmitter in the brain, thereby leading to pleasurable sensations.¹⁸ In other areas of the body, inhibition of the presynaptic reuptake of norepinephrine and dopamine produces a physiologic state similar to that when a sympathomimetic drug is administered.⁷ The adverse effects of chronic abuse of cocaine on the cardiovascular system are, therefore, readily understood.^{19,20}

In the gastrointestinal system, cocaine causes an increased local concentration of adrenergic neurotransmitters in the enteric ganglia, sympathetic neuronal projections to the intestinal epithelium, and the walls of mesenteric blood vessels.⁸ As local increases in norepinephrine occur and

stimulate alpha-adrenergic receptors, there is increased vasoconstriction throughout the gastrointestinal tract.

Both ischemic enteritis and colitis secondary to cocaine abuse have been reported in the surgical literature since 1985.²¹⁻²⁷ The segmental areas of ischemia noted at the time of laparotomy or endoscopy have resolved without resection in some patients. There has been, however, at least one case in which a patient who chronically abused crack developed necrosis of the midgut, mucosal necrosis extending from the midgut to the pyloric antrum, and widespread necrosis and infarcts of the liver and spleen, respectively.⁹

The exact etiology of gastroduodenal perforations after smoking crack cocaine is unknown. Theories proposed or reviewed in previously published papers on the topic have included the following:

1. Focal ischemia¹³
2. Effects on gastric motility¹⁴
3. Increased air swallowing and increased intraabdominal pressure caused by breath holding¹⁵
4. *In situ* vascular thrombosis secondary to cocaine's effects on platelets^{16,28}
5. Increased concentration of ACTH and corticosterone (in rats) with further inhibition of the reuptake of adrenergic neurotransmitters.^{16,29,30}

The most appealing theories to date have been those emphasizing focal vasoconstriction and *in situ* thrombosis. Cocaine has a serum half-life of 45 to 90 minutes, so the relatively early onset of symptoms in patients with cocaine-related perforations of the juxtapyloric area in this series (2 to 4 hours) is readily explained on an ischemic basis. Unfortunately, the theory of acute ischemia is not as acceptable when considering the one patient in this series (no *H. pylori*) and the three patients in the series from Lee et al¹³ whose symptoms started 24 to 60 hours after the use of crack cocaine. And, as stated in the recent publication by Sharma et al,¹⁶ the temporal association between the use of crack cocaine and perforations of the gastroduodenal area "does not confirm a cause-effect relationship."

The 80% incidence of *H. pylori* in the five biopsies of antral mucosa in the later period of the series was a surprise to us. To perform the biopsy, one must obtain an otolaryngologic biopsy instrument and enlarge the site of perforation to allow passage of the instrument proximally into the gastric antrum. With a 2- to 3-mm juxtapyloric perforation that would be easily closed with a viable omental patch, enlarging the perforation and waiting for the instrument to be obtained are not appealing during an emergency operation. And, to avoid errors in transfer of the specimen to the laboratory, it is most helpful to have the CLOtest immediately available in the operating room.

In retrospect, the high incidence of *Helicobacter* in the small number of antral biopsies in this series may be a logical finding. This gram-negative rod is now thought to infect half the population of the world.³¹ In some countries with less-than-hygienic conditions, the seropositivity rate

for *Helicobacter* in children is 70% to 75%.³² In young people living in economically depressed urban areas of the United States, sharing glass pipes on a daily basis, and using an illicit drug that impairs the function of macrophages, a high incidence of *H. pylori* should be found.^{6,31} The unanswered question from the small subset of patients in this series with documented *Helicobacter* infection is whether this predisposes the crack cocaine addict to gastroduodenal perforations.

Appropriate surgical therapy for small juxtapyloric gastroduodenal perforations thought to be related to smoking crack cocaine is the use of an omental patch closure. When a prior history of gastroduodenal ulcer disease is obtained, the addition of a definitive ulcer procedure should be considered for several reasons. The population of crack cocaine addicts with perforations is typically 8 to 10 years younger than the usual patient group with acid-peptic-*Helicobacter* perforations.³³⁻³⁵ This group is unlikely to comply with short- or long-term medical therapy or to complete a program in drug rehabilitation. A parietal cell vagotomy (as was performed in two patients in this series) would be an appropriate additional procedure when the perforated ulcer is juxtapyloric or duodenal, small, and readily covered with an omental patch. When the perforated ulcer is large or located in an area not readily amenable to omental patch closure (*i.e.*, gastric ulcer), gastrectomy with or without truncal vagotomy is appropriate in the hemodynamically normal patient. This is recommended whether a history of ulcers is present or not.^{33,35} Finally, an alternative approach is the use of laparoscopic patching of the perforated ulcer, as reported by Arrillaga et al.³⁶

At the time of discharge, it is unclear whether the patient should be instructed to take short-term H₂-receptor blockers or omeprazole, the proton pump inhibitor, as the relation of gastric acid to these perforations is unknown. In our practice, either agent is routinely prescribed, although it is unlikely to be taken by the patient. When an infection with *H. pylori* has been documented on the biopsy of the antral mucosa, the patient is prescribed omeprazole, clarithromycin, and metronidazole or bismuth subsalicylate for 1 week.³⁷

CONCLUSION

From a review of 50 patients with crack cocaine-related juxtapyloric gastrointestinal perforations treated at one urban public hospital, the following conclusions can be drawn:

1. These perforations occur in a predominately male population of drug addicts who are 8 to 10 years younger than the usual group of patients with pyloroduodenal perforations.
2. The perforations are small and most commonly located in the juxtapyloric area.
3. Omental patch closure is the most appropriate therapy when no prior history of ulcer disease is known.

4. Infection with *H. pylori* may be a contributing factor to these perforations in this population of addicts.

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Discussion

DR. TALMADGE A. BOWDEN (Augusta, Georgia): In 1988, I had the privilege to patch a perforated prepyloric ulcer in a young male who had had no prior history of peptic ulcer disease, but he did admit to using this new drug—at least in the Augusta area—called crack cocaine.

This form of fairly pure cocaine is so named because of the crackling noise that occurs when you heat it and vaporize it. The addicts tell me it sounds similar to milk being poured over Rice Crispies.

The patient did well, but as in this series that you have just been listening to, was lost to follow-up, never keeping his initial post-operative clinic appointment.

Since that time, I have seen in the three hospitals that comprise the teaching facilities for the Medical College of Georgia, three to five patients a year that fit this profile of crack cocaine-associated perforations of the distal stomach or proximal duodenum. There are probably others that we have missed, but sometimes, as Dr. Griffen indicated in his address yesterday, our history-taking may not always be precise, and our patients may not be as honest as we would expect them to be.

Is there an epidemic of this process? The authors indicate by both the title of their manuscript and their experience with 50 patients over a 3-year period, the Medline didn't fail me; I could only locate reports of perhaps 63 cases reported in this decade in the American literature. So for my own edification and perhaps that of the authors, could I see a show of hands from those in the audience who have seen patients with cocaine-associated perforations of the GI tract?

Gosh. That's probably a fourth. Well, clearly, we do have a new

problem in gastroduodenal disease. This problem, which can also give us new opportunities, may shed new light on the exact pathophysiology of peptic ulcer disease, the understanding of which has been for me over the last 30 years like peering in a mirror dimly.

Crack cocaine may provide us with a different animal model for studying, redefining, and treating peptic disease, so I would ask the authors, have you taken this problem to the lab?

The authors have studied the incidence of the presence of *Helicobacter pylori* in five of their patients, finding the organism present in four. The implication of this observation, the role of finding the cause of a perforation, is not clear, since *Helicobacter pylori* has a very significant gastric presence and may or may not cause disease.

I would like to know more about the cellular and vascular conditions that are present in the tissue involved in the perforation, which leads me to another question. Would the authors consider taking a #15 blade knife and shaving off a little bit of the tissue for histological EM, histochemical, or bacteriological studies?

Full-thickness biopsy of perforated gastric ulcers prior to closure of patch is a traditional method, which carries essentially no added morbidity. This simple biopsy may tell us if ischemia indeed has the influence on the etiology of this process that has been postulated.

In two of the 50 patients, the authors chose to add a parietal cell vagotomy because of "prior history of ulcer disease." The manuscript does not give the details of this prior history, but that probably does not matter, because I want to stress that I feel that parietal cell vagotomy is inappropriate in the setting where ischemia may be the etiology of or at least a contributing factor in the development of the perforation.

One of the few complications that we have had with parietal cell vagotomy is the occasional occurrence of lesser curve necrosis, probably on the basis of ischemia. To me, doing a parietal cell vagotomy in a potentially ischemic stomach may add not only insult to injury but injury to injury.

In addition, naturally verbal gastroenterologists would submit that even the noncocaine-associated perforated peptic ulcers do not need an acid-reducing operation in this era of omeprazole and antibiotics, which many of them feel do a better job of reducing ulcer recurrence than does surgery.

Dr. Feliciano, you posed this very question in the *Surgical Clinics of North America* in 1992. What is your answer in 1998? And have you seen any recurrences in your rather large patient population?

Treating gastroduodenal perforations with an omental patch is straightforward, simple, and success can be expected most of the time. There is a small but ever-growing body of literature that indicates that patching perforations laparoscopically may be at least equal to, if not better than, the open method. You did all of your operations by the traditional open method. Did you ever consider laparoscopic approach, and would you consider that now?

Finally, I believe you may have the largest single worldwide institutional experience with crack cocaine-associated gastroduodenal perforations. I believe that provides you with a major opportunity to guide the rest of us in the best short- and long-term management of this problem, and we all look forward to hearing more from Atlanta on this subject.

DR. TIMOTHY C. FABIAN (Memphis, Tennessee): We have either not observed or have not recognized this pathology in Memphis.

Either Memphians are more responsible and upstanding than Atlantans or perhaps the physicians are less observant—probably the latter.

I have a few questions. First, what was the total number of gastroduodenal perforations at Grady over this 3-year period? It would be interesting to compare the crack-induced cohort with the presumed more traditional population.

Secondly, would you define the number of perforations in the duodenal portion, as opposed to the pyloric and prepyloric regions? As in the manuscript, it was stated that the majority were in this area, as was stated today.

Third, it was stated that the median interval from smoking to abdominal pain was 2 to 4 hours. I think, as you indicated in your presentation, how reliable do you think is the illicit drug use history in these patients?

My final question is twofold and related to the average 8-day length of stay. In the manuscript, you mentioned that it was the rationale for your institution to have a 5- to 6-day period of nasogastric decompression, and giving the patients nothing by mouth, of course, during this time. The question is, what is the rationale? And, two, do you have an ongoing project with your psychiatry department involving therapy for this patient population?

DR. RICHARD J. FIELD, JR. (Centreville, Mississippi): In looking back, after reading this abstract, over the cases we have had in the last several years, it did occur to me that most of our cases are younger, they have a shorter period of time of ulcer symptoms. We have done only the omentum patch technique. We have not felt it necessary to do any more definitive procedure, because it has been short-term.

I was intrigued also by your use of the biopsy at the time of the operation. Frankly, that hadn't occurred to me, and I wonder if you would comment a little more in depth about this. Do you simply shave a portion of the edge of the perforation? Or do you go across that into the enteral mucosa of the stomach? And in doing so, would you comment, too, about your real reasons for this?

The serum study of *H. pylori* is pretty accurate. Is it worthwhile doing? Do we have a higher yield? And, if so, I'd like to hear your comments on that.

DR. DAVID V. FELICIANO (Closing Discussion): Dr. Bowden, we have not taken this to the lab. I think some of the questions you raised are very valid, and we have considered clinically shaving specimens to get a little bit more information about whether thrombosis is actually the cause. Because, as I hope I made clear in the presentation, nobody really knows if this is the cause. It seems almost peculiar that this particular area, not necessarily an anatomic watershed in terms of blood supply, is really a site that is so commonly affected.

The question about parietal cell vagotomy is an interesting one for me. I, frankly, hadn't thought about it, Joe. I have never seen a lesser curve perforation. I have certainly read about them in England, and the occasional complication occurring in the United States. But at the time when we first started seeing these, I was an aggressive vagotomist and enterectomist. And it seemed to me this was a lesser procedure for these patients.

I will say that we have learned, in my long experience in public hospitals, that young male addicts do not take medication after discharge. And certainly if I saw a patient this week who had a large perforation after cocaine, with or without a previous history,

I would do a vagotomy and enterectomy. And there is actually a patient on the 6B ward at Grady right now, who perforated in 1988, perforated again about 10 days ago, and Grace Rozycki just did the VNA on him, because, I think, again we all thought that was appropriate.

Laparoscopic patching—probably a reflection of many of these patients coming in at night and knowing that, with a small upper midline incision, we can have this operation done in a matter of minutes.

Dr. Fabian, the total number of gastroduodenal perforations is not available in the manuscript, but I will tell you that we feel that we are seeing a ratio of about three cocaine perforations for every one to two duodenal perforations at the present time. So this actually is exceeding our regular ulcer perforation population. Less than five perforations occurred beyond the pylorus, but, Tim, the reason I left this information vague in the manuscript is because I have been doing perforated ulcers for a long time in public hospitals, and I frequently can't tell where they are because of the inflammation. It is hard to tell if they are prepyloric, pyloric channel, immediately postpyloric, or in the bulb, and therefore, the operative notes were simply not clear enough to distinguish beyond what was presented.

Is the timing of presentation in terms of history by the drug addicts reliable? Of course not, but we did ask them, and I present the information to you.

The reason our patients are kept with a nasogastric tube and NPO is because I was heavily influenced in my career in GI surgery by the late George Jordan, a former president of this Association. And when I was in Houston, we had a very strict policy about keeping everybody NPO and keeping a nasogastric tube in for 7 days, because Dr. Jordan strongly felt that the 8% to 10% incidence of in-hospital re-perforations historically reported could be avoided by not feeding these patients until the patch was better seated. In the modern era, it has been incredibly difficult to get residents to do this, even on my own general surgery service, and I have now, slowly but surely, gotten down to about 5 to 6 days. But it does explain the length of stay.

Tim, all of the patients are offered an opportunity to get into counselling for addiction just through verbal communication to them. We don't force them to do this. We don't keep them in the hospital the way some trauma centers do with their alcoholics. And none of them, to my knowledge, has chosen to be rehabilitated.

Dr. Field, the biopsy really demands that you have to enlarge the perforation because, I have to tell you, these are really small, very typical perforations, with some anterior inflammation. And if you want to stick the EENT biopsy instrument through them and angle it up to the antral mucosa, you are torturing yourself a little bit, because you do have to enlarge it. But, again, the success rate of omentopexy suggests that this can easily be done in those patients.