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HEATER PROBE THERMOCOAGULATION AS A SUBSTITUTE FOR SURGICAL INTERVENTION TO ARREST MASSIVE PEPTIC ULCER BLEEDING: A CONTROLLED, PROSPECTIVE ANALYSIS OF 42 CASES

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ABSTRACT

The goal of this study was to compare the effect of heater probe thermocoagulation for massive bleeding of peptic ulcers with a control group. Between March 1992 and August 1995 we used heater probe thermocoagulation endoscopically to treat 42 patients with active UGI bleeding or nonbleeding visible vessels at the base of ulcer craters within 2-3 hours of admission. We also selected 42 patients with active bleeding or nonbleeding visible vessels who did not receive any endoscopic treatment but were instead treated conservatively as the control group.

The energy applied to each of our patients in the heater probe group was 105±22.5 J (mean±SD). Rebleeding occurred within 2-5 days in 2 patients (4.7%) in the heater probe group versus 9 patients (21.4%) in the control group (p=0.05). Mean duration of admission in the heater probe group was 4.3±3.1 days versus 6.9±3.8 days in the control group which was comparable (p= 0.0027).

There were no statistically significant differences between the two groups concerning transfusion requirement and mortality. Heater probe therapy was tolerated by the patients very well and no complications occurred.

Heater probe thermocoagulation is an effective, safe and economical procedure for treating peptic ulcer bleeding.

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INTRODUCTION

Heater probe (HP) therapy involves the use of a probe consisting of a teflon-coated hollow aluminium cylinder with an inner electronic heating coil. It has the advantage of providing tamponade as well as heat for coagulation. Water may be simultaneously injected to clear the field. Power is computer-controlled for a precise regulation of the probe temperature.[1,2]

The probe must be pushed directly and firmly into the bleeding point to effectively tamponade it. Coagulation is produced after three to four pulses. If bleeding does not stop the probe is repositioned and the procedure repeated.[1,3] Its relative ease of operation, portability, and low complication rate make it a valuable technique.[3,4]

The mortality rate for severe peptic ulcer bleeding has remained constant at 6-10% over the past 30 years.[5-7] Patients with peptic ulcer bleeding are almost exclusively of old age. Postoperative complications of severely bleeding peptic ulcers are many; nevertheles, mortality rates can be lowered with better hospital care including a combined medical and surgical approach, as well as the latest endoscopic therapies.[8-10] In an attempt to reduce the mortality rate, several endoscopic hemostatic modalities have been developed over the past decade to prevent further bleeding and thus obviate the need for high risk emergency surgery.[1,10-12] Since 1978, heater probe thermocoagulation (HPT) has been reported as an excellent means of achieving hemostasis.[4-6] It has been proposed as one of the most promising devices in arresting peptic ulcer bleeding.[7-10]

This article presents the results of HPT compared with a conservatively–treated control group (CG).

MATERIALS AND METHODS

Between March 1992 and August 1995 in a prospective study, a total of 42 patients with massive peptic ulcer bleeding received HPT. We also prospectively chose 42 patients with no history of previous surgery at the beginning of the study with active bleeding due to peptic ulcer and no endoscopic treatment as the control group (CG).

Heater probe thermocoagulation (HPT) was done in every patient with massive bleeding from either a gastric or duodenal peptic ulcer in whom the bleeding source (spurting or oozing) or a nonbleeding visible vessel (NBVV) was observed during emergency endoscopic examination after a period of initial resuscitation.

We used an Olympus GIF-1T20 Panendoscope, an Olympus Heater Probe Unit (HPU), and a 3.2 mm probe to treat the bleeding ulcers. During treatment, the distal tip of the probe was applied directly to the bleeding site with moderate force. 20-30 J/pulse for a total of 4 to 6 pulses was applied to the bleeder. The bleeder was observed for 5 minutes. At the end of this time, the bleeder was challenged with maximal water irrigation for 5-15 seconds. If any further hemorrhage occurred, the above procedure was repeated until no more bleeding was seen. The vital signs of the patients were checked regularly during the first 24 hours until they became stable. A nasogastric tube was inserted and maintained for 24 hours after treatment. The hemoglobin and hematocrit levels were checked twice or three times daily in severe cases and at least once daily for other patients and blood transfusions were given if the hemoglobin level dropped to less than 8 g/dL or the hematocrit to less than 20% along with deterioration of vital signs. H_2 receptor blockers (cimetidine 200 mg IV, q6h) were given routinely. The attending physician and surgeons were made aware of the endoscopic findings and HPT treatment of each case. In some patients re-endoscopy was performed 24-72 hours later. If no active bleeding was observed the patient was discharged and then followed up with endoscopy after one to two weeks and also 8 weeks later. H_2 receptor blockers (famotidine 40 mg or ranitidine 150 mg twice daily) were given orally to every patient for at least 8 weeks. Patients with known liver or renal disease, primary malignancy of the upper gastrointestinal tract or any type of malignancy were excluded.

Patients with active bleeding or NBVV in the ulcer craters who did not receive endoscopic hemostasis or did not have any surgery at the beginning of the study and recovered uneventfully during hospitalization served as controls. They received H_2 blockers (famotidine 40 mg or ranitidine 150 mg twice daily) for at least 8 weeks. Their hemoglobin and hematocrit levels were checked every other day for at least a week. Among CG patients, those who rebled severely were hospitalized again and blood transfusions given when necessary. Emergency endoscopy was performed once again, and medical treatment begun conservatively as soon as possible. Four patients required emergency surgery from the beginning to the end of the study.

The chi-square test was used to compare the two groups. A probability value of less than 0.05 was considered significant.

RESULTS

The patient characteristics of HPT and control groups are shown in Table I. Age, sex ratio, number of patients in shock, initial hemoglobin, and location of bleeders were compared in the two groups. In the HPT group we applied 105±22.5 joules to each patient (95% of CI 91.3-118.6). Some needed less energy and in some we used higher energy to stop the bleeding. The average hospital stay was 4.3±3.1 days in the HPT group compared to 6.9±3.8 in the control group (p=0.002). Initial hemostasis was obtained in

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Table I. Patient characteristics of control and study groups.

Characteristic	HPT group	Controls
	(N= 42)	(N= 42)
Age (yr)	48.3±19.8*	49.2±18
Sex (M/F)	29/13	26/16
Shock(%)	20(47.6)	13(30.9)
Mean hemoglobin on admission	8±3.4	10.5±2.7
NSAID** users (%)	7(16.6)	9(21.4)
Location of bleeders		
Esophagus	0	1
Stomach	21	22
Duodenum	20	19
Stoma	1	0
Endoscopic Findings		
Spurting	10	4
Oozing	26	29
NBVV***	6	9
Applied Energy (Joule)	105±22.5	–

*Mean±SD
**Nonsteroidal anti-inflammatory drugs.
***Non-bleeding visible vessel.

Table II. Results of therapy in the HPT group and the outcome of controls

	HPT group	Controls
	(N= 42)	(N= 42)
Technical success (%)	42(100)	–
Rebleeding (%)	2(4.7)	9(21.4)
Retreated (%)	2(4.7)	–
Emergency surgery (%)	1(2.3)	4(9.5)
Hospital mortality (%)	1(2.3)	1(2.3)
Mean units of blood transfused (range)	5.4±4.9, (0-19)	7.6±2.9, (0-12)
Mean duration (range) of admission (days)	4.3±3.1, (0-13)	6.9±3.8, (0-15)

all 42 (100%) HPT patients but 2 (4.7%) developed rebleeding during hospitalization and received a successful HPT procedure for a second time. The results of therapy in the HPT group and in controls are shown in Table II.

In the control group rebleeding occurred in 9 (21.4%) (p= 0.05 and relative risk in favour of HPT 0.35). Four patients in the control group were subjected to emergency surgery, among which one patient died because of postoperative complications of emergency surgery. One of the HPT patients with a traumatized spine, leg fractures and abdominal trauma had torrential duodenal ulcer bleeding and received HPT. Initial hemostasis in this paitent was complete, but he was operated on 5 hours later because of sustained shock. No further bleeding occurred from the

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duodenal ulcer but the patient had visceral lacerations which were repaired, and he survived. An elderly, frail patient who had multiple medical problems died one week after successful HPT because of acute myocardial infarction.

DISCUSSION

Thermal coagulation should be considered for the treatment of UGI bleeding in the presence of a visible vessel at the ulcer crater, and especially in patients with active bleeding. A vissible vessel may be found in any ulcer crater or may be exposed in a Mallory-Weiss tear. It may appear as a frankly protruding artery, or an elevated red dot (sentinel clot), or be densely adherent to a clot sitting within the crater.[12] The risk of rebleeding from a nonbleeding visible vessel approaches 60 percent.[13-15] The heater probe was first described in 1978 by Protell et al.[1] It works by direct conduction of heat (250°C) to the tissue. Successful hemostatic rates are reported to range between 67-95 percent.[17-24,28] No complications have occurred except for a few perforations.[15,24] There is agreement that torrential, oozing bleeding or a nonbleeding protuberant vessel in an ulcer crater is an indication for urgent HPT treatment.[6] The rebleeding rate for patients receiving HPT has ranged from 8-33% in reported series.[10,12,23,28] We obtained a rebleeding rate of 4.7% which is lower than most reports. The reason for this lower rate is not clear. In fact, we applied less energy (105±22.5 J) to the bleeders compared to Chaudari et al,[28] and much less compared to Lin et al.[19] In our study the rebleeding rate in HPT (4.7%) was significantly different compared to the control group (21.4%) (p= 0.05). Hospital mortality in our patients treated with HPT occurred in only one case which was unrelated to peptic ulcer bleeding (myocardial infarction) and occurred one week after successful HPT treatment. Our only mortality in the control group was directly related to peptic ulcer disease (postoperative complication). No significant complication followed HPT endoscopic therapy in our patients.

We conclude that HPT is quite safe and can decrease the rebleeding rate in patients with massive peptic ulcer bleedings, especially if performed by experts. Like other authors,[19,26] we also believe that HPT may become first line therapy for massive peptic ulcer bleeding in the near future.

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