MASSIVE RECTAL BLEEDING FROM A SOLITARY RECTAL ULCER TREATED BY FIBRIN SEALANT

Gazi University Medical School, Departments of Surgery, Pathology*, and Gastroenterology**, Ankara, Turkey
Gazi Medical Journal 2000; 11: 35-38

B. Bülent MENTEŞ, M.D., Ahmet GÖRGÜL**, M.D., Ertan TATLICIOĞLU, M.D. Ayşe DURSUN*, M.D., Mehmet KAPLAN, M.D.,

Şükrü DUMLU**, M.D., Zafer FERAHKÖŞE, M.D.,

SUMMARY: A case of massive rectal bleeding from a solitary rectal ulcer is described. The hemoglobin level fell to 3 g/dl, and rigid sigmoidoscopy revealed a 5-6 mm wide, 1-2 mm deep solitary rectal ulcer, located on the right anterior rectal wall and about 8 cm from the anal verge. Following unsuccessful attempts to control bleeding with endoscopic coagulation, the ulcer cavity was filled with commercially available concentrated fibrinogen by anoscopic route. Prompt cessation of bleeding with a single injection of fibrin adhesive was followed by conservative measure s to facilitate defecation and stop straining, based on the lack of findings of prolapse. Healing of the ulcer without any recurrent bleeding was confirmed by repeat endoscopies. A new therapeutic choice is thus exemplified for the management of bleeding from a solitary rectal ulcer.

Key Words: Solitary Rectal Ulcer, Fibrin Adhesive, Rectal Bleeding.

Following the first description of solitary rectal ulcer (SRU) by Madigan and Morson (1), surgeons and pathologists have been aware of this rare but distressing condition. A solitary persistent ulcer with distinct histological features forms in the rectal wall and may cause pain, tenesinus, mucus discharge and/or bleeding (2). The etiology and pathogenesis of this disease entity is poorly understood. There is often a history of difficulty in defecation, forceful straining and frequent visits to the lavatory. Chronic straining may lead to internal prolapse which further impedes defecation, and this abnormal defecation dynamic is thought to result in recurrent trauma and chronic ulceration (3). Up

to 60 per cent of patients have overt rectal prolapse and are treated by rectopexy (4). If SRU exists without external prolapse, the diagnosis is often delayed and treatment is generally unsatisfactory (5). Conservative measures to facilitate defecation and stop straining also usually fail. Therefore, although rare, SRU without overt prolapse may cause considerable difficulties in treatment.

This report describes a patient with SRU but without external prolapse, in whom massive rectal bleeding was treated by endoscopic fibrin sealant application.



Fig. 1: Endoscopic view demonstrating solitary rectal ulcer with surrounding fibrosis, located on the right anterior rectal wall and about 8 cm from the anal verge.

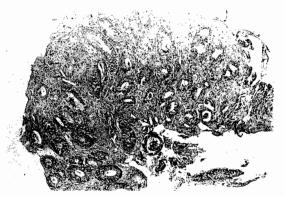


Fig. 2: Histologic view of the biopsy specimen revealed fibromuscular hyperplasia in the lamina propria, reactive hyperplasia of the crypts, and an ongoing healing process, all typical for solitary rectal ulcer.

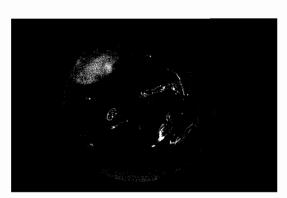


Fig. 3: Plugging of the ulcer cavity with the fibrin adhesive.

CASE REPORT

A 28-year-old male presented with bright red blood per rectum of a week's duration. He had experienced less severe rectal bleeding provoked by defecation for the last two months, but he had attributed these intermittent episodes to hemorrhoidal disease. The medical history was otherwise unremarkable. Upon admission, he was dramatically anemic with tachycardia (118 bpm), hypotension (90/70 mmHg) and orthostatic changes. The initial hemoglobin level was 5 g/dl and hematocrit 19 %. With fluid resuscitation, the



Fig. 4: Videoendoscopic view obtained two weeks after treatment, showing healing of the ulcer with fibrin remnants.

hemoglobin level fell further to 3 g/dl. Rigid sigmoidoscopy revealed a 5-6 mm wide, 1-2 mm deep ulcer with surrounding fibrosis, located on the right anterior rectal wall and about 8 cm from the anal verge (Figure 1). Endoscopic biopsy specimens revealed typical findings of SRU (Figure 2). Five units of whole blood and four units of red packed cells were transfused.

Attempts to control bleeding with endoscopic coagulation were unsuccessful. As the last resort before surgery, we filled the ulcer cavity with commercially available concentrated

fibrinogen (TISSEEL KIT, Eczacibaşı / Baxter, Istanbul) by anoscopic route (Figure 3). The two components of the adhesive were prepared, loaded on the applicator, and injected by anoscopic route to mix and coagulate on the ulcer. This sealing process resulted in prompt cessation of bleeding. A high-fibre diet necessitating daily intake of about 20 g nonstarch polysaccharide was started, and healthy defecation habits were taught to the patient, with particular emphasis on not to strain. Control rectosigmoidoscopy two weeks demonstrated almost perfect healing of the ulcer with no signs of bleeding (Figure 4). Defecography performed three weeks later was completely normal with no findings of prolapse. The patient has been closely followed. If bleeding and/or defecation disorders recur, manometric studies and biofeedback, if necessary, will be performed. There has been no recurrence of bleeding at four months of follow-up.

DISCUSSION

This case report exemplifies a rare but challenging clinical picture related to SRU. The therapeutic difficulty encountered in eliminating bleeding was overcome by utilising a new tool, namely fibrin sealant. Biological surgical adhesives are currently gaining an expanding repertoire of employment in almost every surgical speciality. In United States, autologous fibrin tissue adhesives, made from each patient's own blood and based on ammonium sulfate precipitation or ethanol-freezing precipitation of fibrinogen, are preferred (6). Commercially produced fibrin sealants, manufactured from pooled blood, are naturally more practical and the assembled sealant is nearly ten times stronger than the autologous version. Several such versions of fibrin tissue adhesive have been successfully used in clinical practice as a hemostatic agent or as a tissue sealant to facilitate tissue repair. Throughout the literature on the use of fibrin adhesives, their utilisation in hepatic or splenic surgery as a hemostatic sealants or as a plug to fulfil defects such as anorectal or gastrointestinal fistulas, in particular, has inspired us to try it to control bleeding in a patient with SRU (6, 7-9). This attempt was fortunately successful and provided an excellent example of controlling bleeding from an ulcer by endoscopic

route.

SRU is an uncommon cause of massive gastrointestinal bleeding, and only a few cases have been reflected to the literature (10,11). Such a problem requiring urgent solution naturally causes considerable difficulty in decision making because the etiopathogenesis of SRU is poorly understood. If the condition coexists with rectal prolapse, it is easy to imagine the trauma and the therapy is rather straightforward. Even in some cases without overt external prolapse, evidence of internal prolapse may be detected (12). It is generally believed that chronic straining causes a recurrent traumatic defect on the anterior wall, and if internal prolapse eventually develops, this point also becomes tightly wedged (3, 12). In this respect, Nicholls suggested that combined posterior and anterior rectopexy might be of significant benefit in patients without overt prolapse (12). On the other hand, Kang and coworkers demonstrated that the pathogenesis of SRU is related to factors within the rectal wall itself and is different from that of rectal prolapse, which is the result of external anatomical factors such as intussusception (13). Nevertheless, both entities appear to be similar and may coincide, representing a process of ulceration and repair due to longstanding trauma. Based on the lack of prolapse in our patient and the considerable debate on the relevant therapeutic strategy in the literature, treating massive bleeding was our initial aim. A successful outcome with a single injection of fibrin adhesive was followed by conservative measures to facilitate defecation and stop straining. In addition to symptomatic healing, endoscopic findings were surprisingly good. These short-term results may be attributed to the fibrin sealant, but more patients with longer follow-ups are needed for a more refined conclusion.

To our belief, fibrin adhesives should be available in the surgeon's armament, like suture materials. In selected cases, it may serve as a practical, safe, and physiologic sealant for filling defects/fistulas or hemostasis. In the case of SRU described, both hemostasis and elimination of the tissue defect were provided.

Correspondence to: Dr. B. Bülent MENTEŞ

67. Sok. 16/7 Emek

06510 ANKARA - TÜRKİYE Phone: 312 - 215 35 15,

312 - 214 10 00 / 5719 E-mail: bulentm@ada.net.tr

REFERENCES

- I. Madigan MR, Morson BC. Solitary ulcer of the rectum. Gut 1969; 10: 871-881.
- 2. Mackle EJ, Parks TG. The pathogenesis and pathophysiology of rectal prolapse and solitary rectal ulcer syndrome. Clin Gastroenterol 1986; 15: 985-1002.
- 3. Womack NR, Williams NS, Holmfield JHM, Morrison JFB. Pressure and prolapse the cause of solitary rectal ulceration. Gut 1987; 28: 1228-1233.
- 4. Halligan S, Nicholls RJ, Bartram CI. Proctographic changes after rectopexy for solitary rectal ulcer syndrome and preoperative predictive factors for a successful outcome. Br J Surg 1995; 82: 314-317.
- 5. Ihre T, Seligson U. Intussusception of the rectum internal prolapse: treatment and results in 90 patients. Dis Colon Rectum 1978; 18: 391-396.
- 6. Cintron JR, Park JJ, Orsay C, Pearl RK, Nelson RL, Abcarian H. Repair of fistula-in-ano using autologous fibrin tissue adhesive. Dis Colon Rectum 1999; 42: 607-613.
- 7. Jung M, Manegold BC, Brands W. Endoscopic therapy of gastrointestinal fistulae with fibrin tissue sealant. In. Waclawiczek HW (ed): Progress in Fibrin Sealing. Berlin: Springer-Verlag. 1989; pp 43-52.
- 8. Terblanche J, Krige E, Bornman PC. Simplified hepatic resection with the use of prolonged vascular inflow occlusion. Arch Surg 1991; 126: 298-301.
- 9. Zachariou Z, Roth H. Splenic surgery in childhood. Pediatr Surg Int 1989; 4: 162-167.
- 10. Horsley J, Humphries TJ. Solitary rectal ulcer as a cause of massive lower gastrointestinal bleeding. Gastrointest Endosc 1985; 31: 53-54.
- 11. Binderow SR, Mayer R, Freed JS. Massive hemorrhage from solitary rectal ulcer: toward a definitive treatment. Mt Sinai J Med 1995; 38: 48-49.
- 12. Nicholls RJ, Simson JNL. Anteroposterior rectopexy in the treatment of solitary rectal ulcer syndrome without overt rectal prolapse. Br J Surg 1986; 73: 222-224.
- 13. Kang YS, Kamm MA, Engel AF, Talbot IC. Pathology of the rectal wall in solitary rectal ulcer and complete rectal prolapse. Gut 1996; 38: 587-590.