To the Editor:

With great interest we read the article by Kurt et al (1), describing the use of Ankaferd Blood Stopper (ABS) for controlling gastrointestinal (GI) bleeding due to various benign lesions refractory to conventional measures. Their study included a total of 26 patients diagnosed with the following: Mallory-Weiss tear (n=1), esophageal lesion (n=1), postbiopsy bleeding (n=4), postpolypectomy bleeding (n=11), Dieulafoy’s lesion (n=2), radiation colitis (n=3), gastric antral vascular ectasia (n=3) and congestive gastropathy (n=1). The successful use of ABS in cases of mild to moderate GI bleeding are increasingly being reported (2-6). The major advantages of ABS as a hemostatic agent are its ease of use and lack of side effects. Its use leads to the rapid formation of a ‘hemostatic ABS web’ composed of a protein network enriched with blood cells – particularly erythrocytes – in a unique but not completely defined mechanism, which is presumed to be very different from the classical hemostatic pathway (2). Although the rapid formation of a coagulum in a mildly bleeding source just after spray application of ABS may provide initial excitement to first time users, in our experience, this effect is not strong or durable in patients with moderate to severe bleeding, nor is this effect supported by what is currently reported in the literature (6,7). Therefore, we would like to comment on the conclusions drawn by Kurt et al (1).

Figure 1) A Diffuse bleeding due to rectal telangiectasias. B Endoscopic application of Ankaferd Blood Stopper via heater-probe catheter. C Greenish-yellow coagulum covering the most of the bleeding area except for a point of oozing (arrow). D One week later, diffuse telangiectasias without bleeding are highly apparent.
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First, the title of the article “…refractory to conventional antihemorrhagic measures” is not concordant with the authors’ results. They reported the use of ABS as a primary therapy in 16 cases (patients 2 to 16, and patient 26) and as an adjunct to hemoclips, sclerotherapy or argon plasma coagulation in 10 cases (patient 1, and patients 17 to 25).

Second, they claimed that ABS use achieved hemostasis in all patients within seconds of endoscopic application, despite their explanations regarding the failure of ABS in two patients with Dieulafoy’s lesions. The successful effect of ABS in mild or, in some cases, moderate bleeding described in the study are consistent with reports in the current literature. However, postbiopsy bleeding or postpolypectomy bleeding are usually self limited and stop spontaneously or with simple irrigation in the majority of cases. They did not define the reason for ABS use in the cases that constituted a large part of their series (15 of 26 patients). To us, the unnecessary overuse of ABS in those cases is costly and causes excessive sludge-like coagulum, which makes it difficult to perform other endoscopic procedures during the same session. On the other hand, as they stated, arterial pressure and powerful blood flow does not permit ABS ingredients to linger for a sufficient time to form a plug in severe or spurting types of bleeding. We previously reported the failure of ABS’ hemostatic effect in four cases of severe arterial bleeding (6), in a severe Mallory-Weiss tear (6) and in one case of tumoral bleeding (4).

Third, Kurt et al recommended the use of ABS for oozing bleeding due to angiodysplasia and radiation colitis based on their six cases without mentioning the final outcome of those cases. We previously described the effect of ABS on bleeding in a case of radiation colitis (8) and, more recently, in a detailed manner in eight cases (7). ABS has a transient hemostatic effect lasting one to eight days in bleeding due to radiation colitis (Figure 1). It may lead to an apparent healing of ulcers; however, it is not useful for the treatment of telangiectasias or as a definitive therapy for bleeding (7). We hypothesize that ABS induces a short-lived hemostasis via an ‘ABS web’ on bleeding areas of radiation colitis by inducing powerful aggregation of erythrocytes; however, this reverses due to ongoing underlying inflammation that is primarily mediated by leukocytes.

Fourth, Kurt et al described the effect of ABS even in patients with disturbed hemostasis without mentioning the degree of defect. Recently, a case of Glanzmann’s thrombopathia with gingival bleeding without a benefit from ABS use was reported (9). Moreover, we had two patients (one with hemophilia and one with warfarin overdose) whose ulcer bleeding did not respond to ABS therapy (unpublished data).

In conclusion, until prospective, randomized controlled trials are conducted, the use of ABS should be restricted to only mild to moderate cases of GI bleeding that are refractory to conventional measures (6,10).

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CONFLICTS OF INTEREST: The author has no conflicts of interest to declare.

REFERENCES