contaminated with gastric contents, and the patient went on to develop multisystem organ failure and eventually died of his illness 5 days after the PEG placement.

There are scant data in the literature on the performance of PEG in patients with ascites,[2] and ascites is listed as an absolute contraindication in a practice guideline that is several years old.[3] The risk of peritonitis after the procedure is low,[4] but there is no guidance on prophylactic antibiotics to prevent this feared but uncommon complication. The modern practice of PEG placement requires prophylactic antibiotics to prevent skin and wound infections. In the particular case in question, antibiotics had no clear effect on the patient's clinical deterioration, which was likely caused by progressive multisystem organ failure and hepatorenal syndrome and was not likely a direct effect of unchecked infection. We recommend caution and conservative management of nutritional support in the patient with massive ascites until further data are available in patients with ascites and PEG placement.

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Ankaferd hemostat for the management of tumoral GI bleeding

To the Editor:

Heller et al[1] brilliantly outlined the "difficult-to-manage" situation of tumoral GI bleeding in their review article in *Gastrointestinal Endoscopy*. The authors mentioned a novel hemostatic agent developed in our country, Ankaferd Blood Stopper (ABS; Ankaferd, Istanbul, Turkey), with several potential applications.[1] We herein would like to share our previous experience[2-4] with ABS in GI bleeding. The authors preferred to classify ABS within the context of a "mechanical method for hemostasis." On the other hand, they correctly pointed out that the unique hemostatic effect of ABS presents itself through the rapid promotion of a protein network that anchors erythrocyte aggregation without a direct effect on coagulation factors or platelets.[1] Therefore, the biological basis of ABS, as acknowledged by the authors, should be considered when its effects on the GI system are being evaluated, instead of just pointing out mechanical actions. ABS-induced formation of a protein network with vital erythroid aggregation covers the entire physiological hemostatic process. Mainly, there are distinct important components of the ABS-induced hemostatic network. Vital erythroid aggregation takes place with the spectrin and ankyrin receptors on the surface of red blood cells. Those proteins and the required adenosine triphosphate bioenergy are included in the ABS protein library. Ankaferd also upregulates the GATA/FOG transcription system affecting erythroid functions. Urotensin II is also an essential component of Ankaferd and represents the link between injured vascular endothelium, adhesive proteins, and active erythroid cells. These concepts have been developed via MALDI-TOF proteomic molecular analyses, cytometric arrays, transcription analysis, and SEM ultrastructural examinations as well as numerous investigations using in vitro and in vivo research settings.[5]

Furthermore, there is preliminary evidence that ABS could decrease tumor vascularization in GI neoplasms. Turhan et al[6] reported topical ABS effects in two patients with distinct tumoral GI bleeding due to gastric and rectal cancer. Tumor neovascularization and angiogenesis, before and after the application of ABS, were measured as tumor microvessel density (MVD) in their study. Topical Ankaferd administration to GI neoplastic tissue resulted in the control of bleeding and decreased tumor vascularization in rectal and gastric cancers. ABS significantly decreased MVD measurements in both of the neoplastic tissues in comparison to the MVDs from the biopsy specimens before ABS administration and in unexposed native neoplastic tissues of the stomach and rectum.

Several hypotheses should be raised to understand the mechanism of action of Ankaferd on tumor tissue. The hemostatic action of ABS is correlated with a reduction of tumor neoangiogenesis. Moreover, there is a close relationship between coagulation factor expression and solid tumor progression, via mechanisms other than angiogenesis. In this present study, Ankaferd affected the levels of activating protein 2, androgen receptor, cyclic AMP response element or activating transcription factor 1, cyclic AMP response element binding protein, E2F1-5, E2F6, early growth response, interferon-stimulated response element, Myc-Max, nuclear factor-1, nuclear factor-kappa B, p53 (protein 53 or tumor protein 53), peroxisome proliferator-activated receptor, SMAD2/3, SP1, TPA response element/activating protein 1, and Yin Yang 1 transcription factors.[7]

These regulator molecules affect distinct steps of cellular proliferation, such as cell cycle regulation, angiogenesis, signal transduction, apoptosis, inflammation, acute phase reaction, immunity, and several metabolic molecular pathways.[5,7] In vivo preclinical models should be constructed to elucidate the effects of Ankaferd on neoplastic tissue as described in previous in vitro studies and in vivo observations in bleeding tumors.

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ERRATUM

In "Treatment of chronic radiation proctitis with cryoablation" by Jason K. Hou et al (Gastrointest Endosc 2011;73:383-9), under the head "Cryoablation protocol," the name of the catheter and company were incorrect. The first sentence should have read, "A decompression tube with ports spanning the distal 15 inches of the tube was inserted rectally over a Savory wire. Cryoablation was performed with a 7F, cryoablation catheter (CSA Medical, Baltimore, Md) placed through the accessory channel of the endoscope under direct endoscopic visualization to approximately 0.5 to 1.0 cm from the tip of the endoscope."