our endoscopic armament for direct lesional hemostasis, through its tamponade effect and vasoconstriction, precisely what one would want to avoid in ischemic organ injury. The risk of perforation is also highest in the esophagus, which notably lacks serosa, an additional layer of protection common to the rest of the gastrointestinal tract. It is for this reason that epinephrine injection should be avoided and, importantly, stent placement should not be performed. Indeed, there have been reports of perforated BE in the setting of stent use [5].

Finally, in asymptomatic patients, repeat endoscopy may be helpful in verifying the normalization of the mucosal lining and excluding occult pathology that could have been masked by diffuse BE on initial presentation. This may be performed a few weeks past therapy and should not delay the patient's discharge from the hospital once stable. Stricture or stenosis formation in AEN, which occurs in over 10% of cases during Stage 2 and 3 of the disease, may have an association with concurrent duodenal pathology [2] and seems to be inversely related to the state of immune compromise in affected patients with diabetes mellitus, malnutrition and malignancy [6]. Stricture or stenosis could be managed with outpatient endoscopic dilatation and antacid therapy, but repeat sessions may be necessary.

In the past decade, AEN has continued to ascend the differential diagnosis ladder in hospitalized patients presenting with upper gastrointestinal hemorrhage, largely because of the increased use of endoscopic procedures. Its prompt recognition and proper management will remain important for decreasing mortality and improving the patient's outcome.

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Authors' reply

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We read Dr. Gurvits' comments on our article [1] with great interest and we would like to thank him for his contribution. In fact, our article was inspired by a case we reported in 2019 of an elderly male with multiple comorbidities who presented with acute esophageal necrosis in association with acute cholecystitis [2]. At this point, the review articles by Gurvits *et al*, which provided the first descriptions of acute esophageal necrosis as a complex disease approximately one decade ago [3,4], were very useful in getting a clear view of this widely unknown but potentially serious disease, allowing us to provide adequate management and, fortunately, the patient evolved favorably. Interestingly, we noticed that more than 100 cases of acute esophageal necrosis in association with diverse etiologies (diseases, procedures, drugs, etc.) have been reported during the last decade since the publication of those articles, providing important information regarding novel aspects of its complex pathophysiology and tips to improve its management. Therefore, our article was an attempt to provide an updated and comprehensive review regarding the pathophysiology, etiology, diagnosis and management of acute esophageal necrosis. Gurvits *et al* made some interesting comments on our review article that deserve some discussion.

The association of some drugs with acute esophageal necrosis is interesting and raises the question whether there is actually a causal relationship or the association is co-incidental. In fact, whereas some drugs could result in acute esophageal necrosis, because their mechanism of action could possibly result in esophageal ischemia or direct esophageal mucosal injury, others apparently could not have such effects and an eventual relation with esophageal necrosis has no biological plausibility.

Despite red blood cell transfusion being an appropriate measure to maintain stable hemoglobin levels, when a patient presents with active esophageal bleeding, it may be necessary to perform hemostatic maneuvers. There are cases where submucosal adrenaline injection or placement of a self-expandable metallic stent were effective, suggesting that these measures could be adequate in this scenario [5-7]. However, this is based on case reports and small case series and therefore the level of evidence is not very high. It is actually possible that adrenaline injection could aggravate esophageal necrosis, given its vasoconstrictive effects, and placement of a stent could result in perforation, as suggested by Gurvits *et al*. Although there is no evidence, perhaps hemospray could be equally effective, but safer. Larger studies would help understand which measures would be the safest and most effective in this scenario.

In conclusion, it appears that acute esophageal necrosis is attracting attention in the medical literature and multiple case reports and small case series have been published in recent years. In fact, since the publication of our review, approximately

a dozen more cases have been reported and more are underway. Recently, a multicenter retrospective study involving 36 patients demonstrated that a white color change on endoscopy, septic condition, high pulse rate, and low hemoglobin and albumin are significantly associated with a poor prognosis [8]. More studies like this, involving a larger number of subjects, will be welcome to elucidate this still largely unknown disease.

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