INTRODUCTION:
Endoscopic submucosal dissection (ESD) is the established therapy for superficial gastrointestinal neoplasms. As the larger ulcers associated with ESD, management of artificial ulcers has become more important. However, the relationship between ulcer healing factors and treatment is still unclear.

AIMS & METHODS:
We aimed to evaluate the ESD-related artificial ulcer reduction ratio as 4 weeks to assess the factors associating with ulcer healing after ESD that may lead to optimal treatments. Between January 2009 and December 2013, a total of 375 lesions fulfilled the expanded criteria for ESD. After exclusion, 328 lesions were divided into two groups based on the ulcer reduction rate and analyzed: Group A, rate <90% and Group B, rate ≥90%. These two groups were compared based on clinicalopathological/endoscopic features, concomitant drugs, and treatments.

RESULTS:
The ulcer reduction rate was significantly correlated with factors related to the ESD procedure, i.e., procedure time, submucosal fibrosis, and exposure of the proper muscle layer, in univariate analysis. Multivariate logistic regression analysis showed that submucosal fibrosis (F2) (p = 0.03; OR, 16.46; 95% CI, 1.31–206.73) and exposure of the proper muscle layer (p = 0.01; OR, 4.27; 95% CI, 2.04–8.92) were statistically significant predictors of delayed healing.

DISCUSSION:
The etiology of artificial ulcers after gastric ESD differs from that of peptic ulcers[1]. The healing process of peptic ulcers requires angiogenesis in the granulation tissue at the ulcer base, together with replication of epithelial cells at the ulcer margin, and subsequent re-establishment of glandular architecture[2]. Meanwhile, artificial ulcers were created iatrogenically under the hypoxic environment by PPI injection and normal mucosal protective mechanisms in the shallow layer of the submucosa. As the inflammation is localized, with minimal damage to the proper muscle layer preserving the remarkable contraction, ESD-induced ulcers rapidly reduce in size for the first few weeks[3]. Therefore, artificial ulcers have a particular healing formation involving strong traction towards the center of the ulcer with a little regenerative mucosa (Figure 1a). On the other hand, persistent electrocautery damage to the proper muscle layer, caused by exploiting severe fibrosis or deeper dissection than assumption, can impair the contraction of the mucosa with zipper-like regenerative epithelium.

CONCLUSION:
This single-center retrospective study indicated that ESD-induced artificial ulcer healing was affected by submucosal fibrosis and exposure of the proper muscle layer, which induced damage to the contraction of the muscle layer.

REFERENCES

We disclosed no conflicts of interest regarding this poster presentation.