Endoscopic submucosal dissection for Barrett’s-associated adenocarcinoma in a patient with decompensated cirrhosis and esophageal varices

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Presently, endoscopic resection is the preferred treatment of choice for patients with Barrett’s dysplasia, especially high-grade dysplasia and even early esophageal adenocarcinoma. However, among the various endoscopic resection techniques, endoscopic submucosal dissection (ESD) may carry a higher risk of adverse events in patients with cirrhosis because of the low platelet count, coagulopathy, and presence of esophageal varices. To date, only small case series of esophageal ESDs, especially for squamous cell carcinoma, have been reported from Asia in patients with cirrhosis, but the included patients were well compensated, with platelet counts >50 k/μL. Here, we present a case of Barrett’s-associated adenocarcinoma in a patient with decompensated cirrhosis, platelet count <50 k/μL, and esophageal varices, who was successfully treated with ESD (Video 1, available online at www.VideoGIE.org).

A 65-year-old woman with a history of Barrett’s esophagus (BE), decompensated alcoholic cirrhosis (Model End-stage Liver Disease score 8, Child Pugh class B) complicated by ascites, esophageal varices, gastric antral vascular ectasia, hepatic encephalopathy, and severe thrombocytopenia underwent surveillance EGD for BE. EGD noted a nodular lesion arising from the BE (C1M3) with a biopsy reading of at least intramucosal cancer. Under EUS, the lesion was not well visualized because of diffuse mucosal edema. Submucosal and extramural varices were seen in the distal part of the esophagus (Fig. 1). No obvious muscle or lymph node involvement was noted. CT of the chest, abdomen, and pelvis with intravenous contrast material showed no evidence of locoregional lymphadenopathy or distant metastasis.

Figure 1. EUS view showing submucosal (yellow arrowhead) and extramural (orange arrowhead) varices in the distal part of the esophagus. The lesion was not well visualized because of diffuse mucosal edema.

Figure 2. EGD view demonstrating a 15- x 10-mm depressed lesion (yellow arrowhead) with disrupted surface pattern in the esophagogastric junction at 28 cm from the incisors.

Figure 3. Underwater examination with narrow-band imaging and near focus showing better delineation of the margin of the lesion.
The case was discussed with a multidisciplinary esophageal cancer team to explore treatment options. A decision to pursue endoscopic resection was made based on the need for accurate tumor staging and the patient’s and family’s preference for minimally invasive treatment over no treatment or surgery. Given the lesion size, and prior biopsy results indicating at least intramucosal cancer, ESD was planned to offer an accurate pathologic diagnosis and curative resection for early BE adenocarcinoma. Although the patient was at high risk for the procedure because of her cirrhosis, her ascites, encephalopathy, and esophageal varices were well controlled with medications. Because of her baseline low platelet count (30 k/μL), she was given avatrombopag, a thrombopoietin receptor agonist, 5 days before the procedure. Her platelet count was 48 k/μL after she received avatrombopag. She received a 1-unit platelet transfusion during the procedure.

The procedure was performed with the patient under general anesthesia. EGD demonstrated a 15- × 10-mm depressed lesion with a disrupted surface pattern in the esophagogastric junction at 28 cm from the incisors (Fig. 2). Underwater examination with narrow-band imaging and near focus was performed to get the best images for examination and to better delineate the margin of the lesion (Fig. 3). Thermocautery markings were placed 5 mm around the lesion edge. The lesion was noted to be hypervascularized. Major oozing and visualized blood vessels were controlled with

Figure 4. A long, tortuous submucosal varix was identified during submucosal dissection.

Figure 5. Mucosal defect after endoscopic submucosal dissection showing no evidence of bleeding or perforation.

Figure 6. Resected specimen after endoscopic submucosal dissection.

Figure 7. Microscopic view of specimen showing abundant uninvolved submucosa, as evidenced by the presence of an esophageal submucosal gland. The luminal surface is at the top of the image. There is squamous epithelium at the upper left of the image. An intramucosal adenocarcinoma is seen extending into the muscularis mucosae (H&E, orig. mag. ×2).

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coagulation forceps, and minor bleeding was treated with a close tip of the DualKnife. A long, tortuous submucosal varix was identified, which was procoagulated with coagulation forceps (Fig. 4). The varix was cut after being coagulated. The lesion was removed in en bloc fashion (Figs. 5 and 6). The total procedural time was 60 minutes. After the procedure, the platelet count remained stable (45,000). She was admitted for observation, and her postprocedural course was uneventful.

Final pathologic examination revealed a T1a esophageal adenocarcinoma, in a background of high-grade columnar epithelial dysplasia. Both lateral and deep margins were noted to be negative (Figs. 7 and 8). No poorly differentiated component or lymphovascular invasion was identified. Curative resection was achieved, and surveillance endoscopy with treatment of remaining BE was recommended.

In conclusion, esophageal ESD is feasible for Barrett’s-associated superficial adenocarcinoma in a decompensated cirrhotic patient with esophageal varices and severe thrombocytopenia. Esophageal varices, portal hypertension, and severe thrombocytopenia are not absolute contraindications for esophageal ESD, if the procedure is performed carefully after proper medical optimization preprocedureally. Extra care should be taken to avoid inadvertent injury to the submucosal varix for successful completion of the procedure.

**DISCLOSURE**

Dr Kalloo is a founding member, equity holder, and consultant for Apollo Endosurgery. Dr Ngamruengphong is a consultant for Boston Scientific. All other authors disclosed no financial relationships.

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