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Rapid growing esophageal adenocarcinoma 1case

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Barrett's esophagus is the risk factor for esophageal cancer but the pathogenesis of Barrett's esophagus and the mechanism of transformation into adenocarcimoma is uncertain. The author sees the rapidly growing esophageal adenocarcinoma, look like Barrett's esophagus and report it. A 83-year-old male vistited our hospital suffering from dysphagia and weight loss 8 months ago. Initial vital sign was 110/70-78-20-36.4. Laboratory data shows Hb 13.0 g/dL, PT(INR)1.0, BUN 21.7 mg/Dl. Endoscopic examination demonstrated 3cm sized tongue like mucosal projection at squamous columnar junction from incisor 34cm(Fig.A) Endoscopic biopsy was done, the result of biopsy was confirmed as chronic inflammation with focal atypical cell and moderate sguamous epithelial dysplasia. No definite mass formation along esophagus on CT. 3 month later, follow up endoscopic biopsy was done, the result of biopsy was confirmed as epithelial hyperplasia and mild dysplasia. (Fig.B) Esophageal stricture was noted but endoscopic passage was possible. 5 month later, he was admitted to our hospital due to vomiting. Endoscopic examination demonstrated submucosal tumor like Buldging lesion with central ulcer from incisior 32cm. Esophageal stricture was noted and endoscopic passage was impossible.(Fig. C) Endoscopic biopsy was done was confirmed was adenocarcinoma, poorly differentiated. Suspicious necrotic nodular lesion in lower thoracic esophagus on CT.(Fig.D) Patient performed cervical esophgogastrostomy with lymph node dissection and proximal gastrectomy and tubogastroplasty. Finally confirmed stage IIIa (T2, N2, M0). When Barrett's esophagus is suspected in endoscopy with alarm signs such as weight loss, dysphagia are present, close observation and regular follow up should be considered

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Switching from TDF and NUC Therapy to TDF Monotherapy in Virologically Suppressed CHB Patients

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Background/aims: It is unclear whether chronic hepatitis B (CHB) patients with antiviral resistance, who achieve a complete virologic response (CVR) with tenofovir disoproxil fumarate (TDF) and nucleoside analogue (NUC) combination therapy, maintain CVR if switched to TDF monotherapy. We investigated the persistence of CVR after cessation of NUC in virologically suppressed antiviral resistant CHB patients using TDF + NUC combination therapy. **Methods:** This study recruited 76 antiviral resistant CHB patients showing CVR on TDF + entecavir (ETV) (n＝52), TDF + lamivudine (LAM; n＝14) and TDF + telbivudine (LdT; n＝10) combination therapy, who were switched to TDF monotherapy as step-down therapy. **Results:** At baseline, 47 patients were male and the median age was 53.0 years (range: 30-78 years); 72.3% cases were hepatitis B e antigen-positive (HBeAg+) and 23.7% were of liver cirrhosis. The median duration of TDF + NUC combination therapy was 20.8 months (range: 3-46 months). At a median follow-up of 24.7 months (range: 9-45 months) after switching to TDF monotherapy, all 76 patients maintained CVR, regardless of the duration of combination therapy and the type of prior NUC and antiviral resistance. Renal dysfunction was not observed during the treatment period. **Conclusions:** The step-down strategy of switching from TDF + NUC combination therapy to TDF monotherapy in virologically suppressed CHB patients with antiviral resistance should be considered.