Introduction:
Esophageal atresia/Tracheoesophageal fistula (EA-TEF) affects 1:2400 births. Expectant management of long-term complications such as strictures, esophageal dysmotility, gastroesophageal reflux disease (GERD) and a risk of Barrett’s esophagus (BE) is increasingly relevant with improved survival rates. Eosinophilic esophagitis (EE) is a chronic inflammatory condition associated with eosinophilic dysphagia which has only rarely been reported in EA patients.

Here we report the case of a young girl with EA diagnosed with EE and BE 4 years after her delayed primary repair.

Case Report:
Our patient LK was born with long gap, Type A, EA and underwent Foker esophageal growth augmentation followed by delayed primary anastomosis at the age of 3 months. Although proton pump inhibitors were prescribed, her mother was non-compliant with administering her medication.

LK developed recurrent anastomotic strictures and required 13 radiological dilatations, mostly within the first year of life. She had a significant background of atopy, having both asthma and eczema, but had no food allergies (this was later confirmed with skin prick testing).

At age 4, she was referred to our center’s multidisciplinary EA-TEF clinic for the first time by her surgeon. By then she had increasing symptoms of dysphagia, regurgitation and food impaction. A barium swallow showed a mild narrowing at level of initial anastomosis with no hold up of contrast.

**Initial gastroscopy** showed white exudate with furrowing and erosions in the distal esophagus.

Histopathology from distal esophageal biopsies showed eosinophils numbering up to 55/HPF proximally and 60/HPF distally. There was also hyalinisation of the subepithelial stroma. H&E 200x.

Follow up gastroscopy after 3 months showed resolution of furrowing, exudates, and erosions. There was however abnormal salmon coloured mucosa suggestive of BE arising above the lower esophageal sphincter (LES) and extending for 1 cm proximally at the 12, 6, and 9 o’clock positions not previously observed. Biopsies confirmed intestinal metaplasia. The number of intraepithelial eosinophils had reduced proximally to 10/HPF and distally to 80/HPF.

CONCLUSION:
1. Studies suggest that up to 11% of adults with EA-TEF develop BE with an estimated lag time of 10 years between surgical correction and metaplastic changes. An ongoing prospective study of 15-19 year olds found that only 1 in 88 had developed intestinal metaplasia. Other studies report varying prevalence of intestinal metaplasia in children with a mean onset in their teens.
2. Our patient is the youngest in reported literature with BE (intestinal metaplasia +ve) changes post EA repair.
3. EE has only rarely been reported in patients with concomitant BE. The youngest patients reported were aged 10 and 14 years at diagnosis making our patient the youngest reported thus far. It is unclear in our patient however, if the BE was secondary to untreated GERD or EE or both.
4. Prospective multicentre studies are required to determine if EE is an independent risk factor for the development of BE.
5. The diagnosis of BE in our 4 year old EA patient highlights the need for early and regular endoscopic surveillance in this vulnerable patient population.

References: